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FAMILIAL LUMBO-SACRAL SYRINGOMYELIA AND THE SIGNIFICANCE OF DEVELOPMENTAL ERRORS OF THE SPINAL CORD AND COLUMN.

By MURRAY JACKSON,
Royal Prince Alfred Hospital, Sydney.

THE observation of a family with a high incidence of sensory disorders of the lower limbs associated with trophic ulceration of the feet and *spina bifida occulta* stimulated a search into the nature of this disease.

In 1903, Fuchs[1] used the term "myelodysplasia" to describe an abnormal development of the spinal cord. Since that time a new concept has arisen regarding the significance of developmental errors of the spinal cord and column. Recent work is suggestive that these errors are by no means uncommon and that they may (i) produce various disorders, such as lumbo-sacral syringomyelia, which is described in this paper; (ii) be unproductive of symptoms and recognizable only by the radiological detection of a *spina bifida occulta*; (iii) be the result of an inherited type of constitution—*status dysraphicus*, to which type some 17% of the population belong. Evidence in favour of these conclusions will be discussed later in this paper.

The cases presented here afford a striking example of the rare neurological disorders which may result from anomalous cord development.

Familial Lumbo-Sacral Syringomyelia.

Less than one hundred cases of this rare disease had been reported prior to 1942. The largest series was that of Van Epps and Kerr,[2] who observed ten cases in three generations.

The family reported on here have lived for eighty years in the Goulburn district of New South Wales. Their medical history was uncovered when the relationship was traced of two patients in the Royal Prince Alfred Hospital suffering from trophic ulceration of the feet.

Of the twenty-six cases occurring in four generations (see Figure I), eight were personally investigated. From these cases and from the reports concerning other members of the family, the natural history of the disease can be described. In this family it follows essentially the same course as in others reported. A significant difference is the common occurrence of sensory changes in the hands, a phenomenon which does not seem to have been reported previously.

The patient usually suffers from calloused feet from an early age. The age of onset of symptoms varies greatly. The average age in this series is thirty-five years, with limits of fifteen and forty-seven years.

The first sign is usually the appearance of an ulcer beneath the ball of the foot or great toe. In many cases this is the result of trauma. Loss of sensibility in the feet precedes the appearance of the ulcer, which is painless, but subjective appreciation of the sensory change is not usual before the ulcer appears. The sensory loss may be of dissociated type.

The ulcers heal with adequate rest, but break down again when the patient starts to walk. Bony changes soon develop in the feet. They consist of a generalized osteoporosis with bone absorption commencing at the metatarsal heads. The process extends to the phalanges which eventually disappear, with resultant shortening of the toes.

There is gross disorganization of joints, particularly the metatarso-phalangeal, similar to that seen in tabes. The metatarsal heads become spindle-shaped and eventually disappear. Pieces of bone are discharged through the ulcers from time to time, and secondary osteomyelitis is common. Coincident with these bony changes, the foot undergoes a longitudinal shortening, due to bone absorption, and eventually becomes merely a stump with only remnants of toes. These striking bone changes are remarkably constant in nature. Motor disturbances are minimal, and can be attributed to disuse atrophy of muscles.

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As well as trophic and sensory changes, vasomotor changes may occur. These are demonstrated by a reduction in flares produced by the intradermal injection of histamine. This was seen in the three cases to which the test was applied, and it conforms to results recently reported. Differences in temperature in the lower limbs may also occur.

The disease is usually painless, except when infection flares up causing considerable pain. The soles of the feet eventually become completely anæsthetic.

Abnormalities of the spinal column are common, and among the eight patients examined *spina bifida occulta* occurred in five cases, and lumbar ribs as well in two cases. Apart from the foot lesions, the general health is surprisingly good. There are no other abnormal nervous findings, no enuresis and no abnormality in sex life.

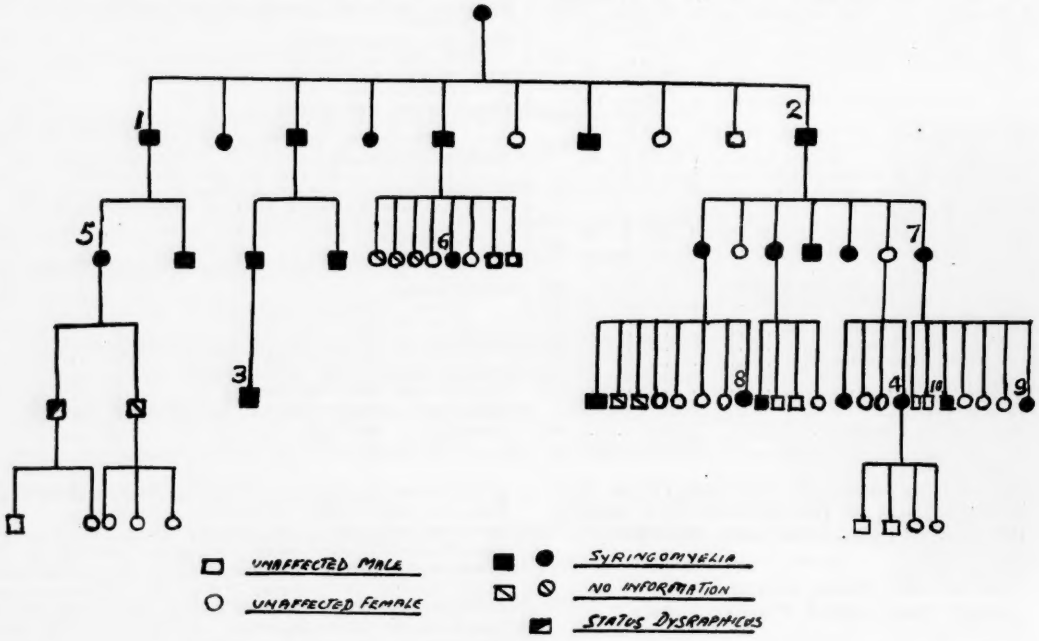

FIGURE I.

Chart showing 26 cases of familial lumbo-sacral syringomyelia occurring in four generations of one family.

The course of the disease is very slowly progressive, the ulceration rarely extends above the ankle, and the expectation of life does not seem to be shortened.

The case of Chris E. is typical of an advanced case of the disease.

CASE I.—Chris E., a male, aged thirty-three years, first noticed symptoms at the age of fifteen years when he began to lose thermal sensibility in his feet. At the age of seventeen years he burnt his right foot when walking on hot ashes—the burn was painless. An ulcer formed at the site of the burn, and healed only when the underlying bone was curetted. At the age of eighteen years the ulcer broke down again, and he came to the Royal Prince Alfred Hospital. When examined he was found to have a *spina bifida occulta* consisting of non-fusion of the laminæ of the first sacral vertebra. There were anæsthesia of the soles of the feet and impairment of thermal and pain sensibility below the knees. A large chronic and painless ulcer was situated on the ball of the right foot.

In 1931 a laminectomy was performed. Some fatty masses were found between the skin and dura, and removed. The dura appeared abnormal, in that it terminated at a higher level than usual. The *cauda equina* appeared normal, and the cord was not fixed. There was no appreciable improvement in the condition during the next two years. At this stage it was noticed that the legs were definitely colder than the arms. In 1933 a bilateral lumbar sympathectomy was performed. The ulcers persisted, and in 1935 the heads of the first and second right metatarsals were excised in an effort to eradicate bone infection. Since 1935 this patient has been treated in hospital many times for ulcers on the soles of both feet. Treatment consisted of rest and local stimulation, and usually resulted in healing of the ulcers. As soon as the patient left his bed the ulcers began to break down again.

When examined at the Royal Prince Alfred Hospital in 1945, his feet were seen to be greatly deformed. There was gross longitudinal shortening of the feet and toes. There was a large ulcer on the ball of each foot, a third beneath the base of the fifth right metatarsal, and gross callosity of the skin of the feet (Figure II). General examination revealed no abnormality of lungs, heart or alimentary system. Examination of the nervous system revealed slight motor weakness of the legs; all reflexes were intact. Sensory changes were gross, and consisted of complete anæsthesia of the soles of the feet and dissociated anæsthesia up to the knees (Figure III). Cranial nerve functions were normal. Examination of the vascular supply of the lower limbs revealed a normal reaction to elevation and depression of the legs. The *dorsalis pedis* and posterior tibial arteries were pulsating normally. Flares produced by the intradermal injection of histamine were greatly reduced below the knees (Figure IV). Skin temperatures in the legs were found to decrease peripherally (Figure V). The result of the blood count was within normal limits. The results of blood Wassermann and Kline tests were negative. The blood urea, uric acid and glucose content was within normal limits. The cerebro-spinal fluid was apparently normal.

Radiological examination of the feet showed gross osteoporosis with absorption of metatarsal heads and phalanges, and disorganization of the joints of the feet. Radiological examination of the hands and of the cervical part of the spine revealed no abnormality.

When he was examined at the Royal Prince Alfred Hospital in January, 1947, it was noticed that there was a definite impairment in thermal sensibility in the palm of the left hand.

In early cases there is no sensory impairment in the hands. As the ulceration and bony absorption progress, the sensory loss extends upwards to the knees, and finally affects the hands.

CASE II.—Mabel C., aged thirty-three years, had no trouble with her feet until two years ago, when the skin of the

medial surface of the great toe of the right foot sloughed, leaving an ulcer. This ulcer has not yet healed, and is painful when she has walked any distance. Examination showed a healthy, well-built woman, whose feet were not deformed. An ulcer was seen on the medial surface of the great toe of the right foot. Examination of sensory function revealed complete dissociation of anæsthesia in the toe affected. Below the ankles, tactile sensation was normal, pain sensation diminished and thermal sensation absent. There was no impairment of sensation in the hands. Radiological examination of the feet revealed early absorption of the terminal phalanges; no bony abnormality was found in the lumbo-sacral part of the spine.

CASE III.—Charles C., aged forty-two years, experienced swelling of the toes of his right foot at the age of thirty-two years, and the foot became slightly painful. The nails of the second, third, fourth and fifth toes became detached painlessly. Gradually these toes shortened. Small blisters began to form over the heads of the metatarsals. These blisters would appear, break down and discharge pus, and heal very slowly. For a few days before the breaking of these blisters, the patient would become hot and feverish, and the right foot would swell. With the discharge of pus, the symptoms would abate. The left foot has not been affected.

Examination revealed a robust man, whose feet were not deformed. There was a small ulcer on the medial surface of the great toe of the right foot. Sensory examination

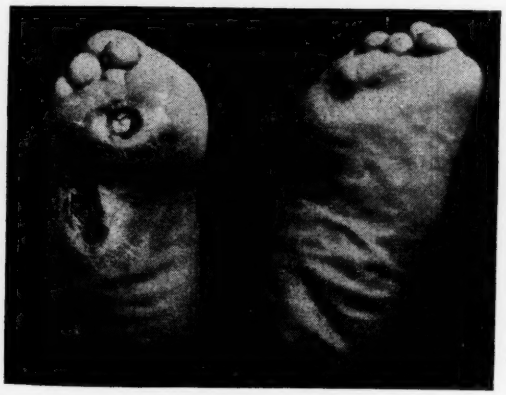

FIGURE II.
Chris. E. Involvement of feet.

showed complete dissociation of anæsthesia in both legs, up to mid-calf, with impairment of pain and thermal sensation up to the knee. There were no other positive nervous signs.

Radiological examination of the right foot showed typical early changes. The middle and terminal phalanges of the second to the fifth toes were entirely absorbed, and the head of the fifth metatarsal had undergone early change. The middle and terminal phalanges of the left foot were in the process of absorption. On examination of the lumbo-sacral part of the spine a rudimentary rib on the left transverse process of the first lumbar vertebra was seen, but there was no evidence of *spina bifida occulta*.

Some of the patients had been subjected to operations which aimed at correcting the disturbed dynamics of the bones of the feet. Daphne G. derived no benefit from such an operation.

CASE IV.—Daphne G., aged forty-three years, developed swelling of the right foot at the age of twenty-one years, and an ulcer formed on the sole of the foot beneath the head of the fourth metatarsal. This ulcer was painful, and healed only when she went to bed for a week. As soon as she started to walk again the ulcer broke down. This process of healing and relapsing has continued up to the present. In 1937, at the age of thirty-four years, she underwent an operation consisting of correction of bilateral *hallux valgus*, arthrodesis of the metatarso-phalangeal joints of the great and first toes of the right foot, and tenotomy of the right *tendo Achillis*. Since that time, the condition has progressed more rapidly. The right foot and toes have become shortened, and the third toe of the left foot is affected. This toe is swollen and the nail is becoming detached. There has been no ulcer on the left foot as yet. The patient suffers from intermittent attacks of fever, associated with cellulitis of the right leg and inguinal adenitis. At other times the right foot sweats profusely for no apparent reason. She has noticed the loss of thermal sensibility in the hands.

Examination revealed a deformed right foot. The toes were short and there were two ulcers on the ball of the foot. The left foot appeared normal except for the third toe, which was swollen, and the nail, which was loose and gryphotic. There was total anæsthesia of the sole of the right foot, and dissociated anæsthesia up to mid-calf of both legs, reaching a higher level in the right leg. Above this was seen a band of impairment of pain and thermal sensation. Both hands were affected with dissociated anæsthesia.

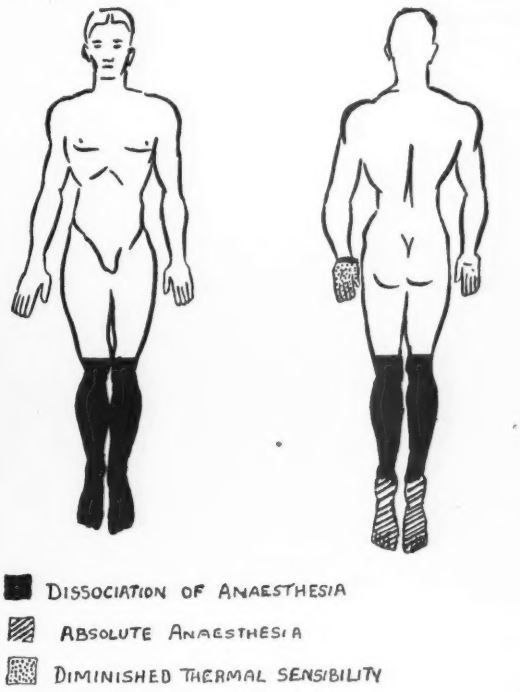

FIGURE III.
Chris. E. Sensory disturbances.

Radiological examination of the right foot showed well-established changes. The shaft of the fifth metatarsal was narrow, and the terminal phalanges had undergone advanced absorption. Early changes were seen in the terminal phalanges of the left foot. On examination of the lumbo-sacral part of the spine *spina bifida occulta* of the first sacral vertebra was seen, with a rudimentary rib on the left transverse process of the first lumbar vertebra.

Gertrude M. derived no benefit from an operation designed to correct her bilateral *hallux valgus*.

CASE V.—Gertrude M., aged fifty-three years, first had symptoms in 1938 at the age of forty-five years. A nail from her shoe pierced the great toe of her left foot, forming an ulcer, which discharged pus and did not heal. A short time later, other ulcers appeared on the toes of the left foot. The toes rapidly shortened, and the nails became detached. At this stage she underwent an operation for correction of bilateral *hallux valgus*. Shortly afterwards, two large ulcers appeared on the sole of the left foot, and discharged pus and small pieces of bone. In 1940 the right foot became involved. An ulcer appeared on the plantar surface of the second toe, which was amputated. In 1941 the left leg became swollen and has remained so. The feet are painful at times, especially prior to the discharge of copious pus by the ulcers. These bouts of pain are associated with fever and cellulitis of the left leg. Since 1942 she has noticed great impairment of tactile sensibility in the hands.

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Examination showed gross swelling of the left leg and deformity of the feet, particularly the left. This foot was shortened; the second toe was absent, and the remainder were very small. There was much verrucose hyperkeratosis of the sole of the foot and great toe, and a large ulcer on the sole of the foot. The right foot was less deformed and had an ulcer on the sole. Sensory function was impaired in the legs and hands. In the legs anæsthesia extended to mid-calf, and there was impairment of all sensation up to the knees, the changes reaching a higher level in the left leg. There was loss of tactile sensibility in the fingers.

Radiological examination revealed that in this case the changes were more advanced in the left foot; there was widespread bone absorption and the metatarsal heads and the phalanges of the second and third toes were completely absorbed. The same changes were present in the right foot to a lesser degree. On examination of the lumbo-sacral part of the spine a *spina bifida occulta* of the first sacral segment was revealed.

The other three cases were essentially similar to Case I, and will not be described in detail.

FIGURE IV.

Chris. E. Reaction to intradermal histamine injection.

The Nature of the Disease.

This disease has been named at various times *"trophopathia pedis myelodysplastica"*,[3] "familial trophoneurosis"[4] and "familial lumbo-sacral syringomyelia".[2] This last name is probably the least objectionable, although cavitation in the lumbo-sacral segment of the cord may not necessarily be present.

That the disease is an inherited developmental defect of the spinal cord and column is shown by the familial incidence and the association of errors in development of the spinal column—*spina bifida occulta* in five cases of this series and lumbar ribs in two of these cases.

Normal Development of the Spinal Cord and Column.

Development of the spinal cord and column commences with the formation of the neural plate in the embryonic ectoderm in the dorsal median region (Figure VI).

The neural groove now appears and the neural folds form and fuse. From the neural crest area are derived the posterior root ganglia and sympathetic ganglia. Fusion of the neural folds forms the neural tube. The notochord forms ventral to the neural tube as a condensation of mesoderm (Figure VII).

The mesodermal somites proliferate and cut the tube off from the surface ectoderm. The developing notochord forms the vertebral body, and sends off costal and neural processes, precursors of the ribs and laminæ (Figure VIII).

The cord differentiates into marginal, mantle and ependymal zones, and the laminæ fuse. The spinous processes are formed at about the tenth or twelfth week (Figure IX).

Closure of the neural folds is completed last in the lumbar and cervical regions, where the neuropore and caudopore persist for some time. Recent work by Leveuf[5] has shown that the cord development caudal to the caudopore differs significantly. After closure of the caudopore the posterior part of the cord develops as a solid rod of cells in the neuroectoderm. The central canal appears in this.

In 40% of embryos multiple canals appear in this region, and the final central canal is formed either by coalescence of the multiple canals or by the displacement of all but one to the surface. Disorder of this "secondary development" of the cord accounts for anomalies such as double canal and reduplication of the cord or diastematomyelia.

After the third month of intrauterine life, the column grows more quickly than the cord, with a resultant relative upward migration of the cord.

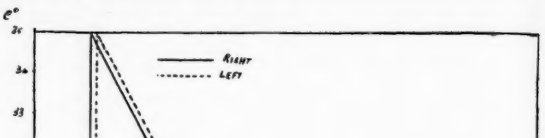

FIGURE V.

Chris. E. Skin temperature readings by dermotherm. (There was considerable inflammation of the right great toe.)

The *filum terminale* develops early by obliteration of the central canal in the lowest coccygeal segments, which are drawn out into a thread by the upward migration of the cord, being attached caudally to the skin overlying the last coccygeal segment.

Abnormal Development of the Spinal Cord and Column.

The frequency of anomalies of closure of the neural folds in man and the vertebrates indicates that this mechanism is an extremely delicate one.

From the study of the normal development it can be seen that disorders of closure can vary in degree from complete failure of fusion, producing a myelocele, to a slight bony defect, such as a bifid spinous process.

The clinical manifestations of this disordered or "dysraphic" state may appear in the somatic ectodermal derivatives, the mesodermal derivatives, or the neuroectodermal derivatives.

The following table (Table I) adapted from Lichtenstein[6] classifies the possible resultant defects:

There may be inclusions of mesodermal tissue in the lower canal.

Most of these defects are well known, but the clinical significance of disorders of neuroectodermal development has been little appreciated until recently.

Myelodysplasia.

The term myelodysplasia was used originally by Fuchs[1] in 1903 to describe the neural expression of spinal dysraphism. As seen from the foregoing, it varies in degree

from complete myelocele to cases in which there may or may not be a *spina bifida occulta* to indicate that the underlying cord is abnormal.

Clinical Types of Myelodysplasia.

Myelodysplasia is probably more common than is generally realized. The following classification of clinical types is suggested by case reports and personal observations: group I: extreme degrees of myelodysplasia, such as myelocele and other forms of *spina bifida aperta*; group II: lumbo-sacral syringomyelia; group III: primary cervical syringomyelia; group IV: hydromyelia and some intramedullary tumours of the cord; group V: some cases

TABLE I.

Embryonic Tissue.	Defect.	Resultant Defect.
Somatic ectoderm	Cutaneous.	Cutaneous defect. Hypertrichosis. Hypoplasia of skin. Congenital dermal sinuses.
Mesodermal ..	Vertebral.	Degrees of bony abnormality.
	Dural.	Non-fusion of dura.
Neuroectodermal	Neural tube.	Myelodysplasia.
	Neural crest.	Ectopia of spinal ganglia.

of enuresis and nervous disturbances of the lower limbs, usually diagnosed as *spina bifida occulta*; group VI: some cases of vasomotor sensory and trophic disturbances of the limbs, the *formes frustes* of syringomyelia. This classification is probably incomplete, and doubtless other types will be added when the condition is more widely recognized. The fundamental pathology is common to all types, and intermediate and combined forms may be seen, such as syringomyelia coexisting with intramedullary tumour. Acrocyanosis and congenital talipes may eventually be included.

Full discussion of these clinical types will be the subject of a later report.

In all these forms some anomaly of the vertebral column is common, particularly *spina bifida occulta*. Nevertheless a normal spinal column does not necessarily indicate a normally developed spinal cord.

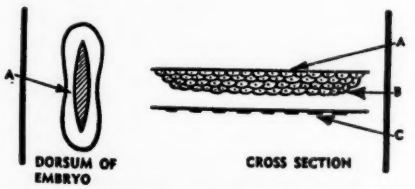

FIGURE VI.

Pathogenesis of Myelodysplasia.

The primary error of development in myelodysplasia lies in the closure of the neural tube and the formation of the dorsal median raphe. If the tube remains open the serious forms of *spina bifida aperta* result. Faulty closure may give rise to hydromyelia, or to pseudoduplication of the cord. The other forms of myelodysplasia arise as a result of inclusion in the cord of rests of primitive medullary epithelium.

The final clinical manifestations depend on the changes undergone by these rests, and on their extent and location. In recent works, Lichenstein,[6] Tamaki, Lubin[7] and others have described the formation and evolution of these cell rests.

Pathological material has been insufficient to permit description of the exact pathogenesis of each clinical form of myelodysplasia. The changes undergone by the medullary rests consist in differentiation of the primitive medullary epithelium into ependyma and glia. To this process the term "primary ependymal gliosis" is applied. The glia surrounding the ependymal cells tends to proliferate and, by virtue of its poor blood supply, may undergo necrosis.

It is here that the syringomyelic cavity arises. If the process continues, the cavity may grow and be partly or completely lined by ependyma.

Dissociated anæsthesia will occur if the pathological process interrupts the fibres crossing in the anterior white commissure.

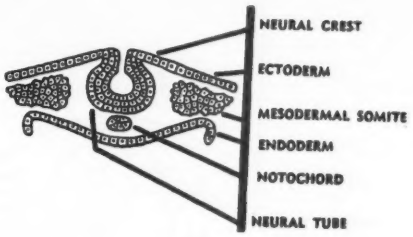

FIGURE VII.

Other changes may occur. The ependymal cells may proliferate and may eventually give rise to an ependymal neoplasm. The ependymal cells may degenerate and undergo gliosis. Again, the area may become the site of repeated hæmorrhages.

These pathological changes have been well established by numerous observations. Cox[8] described a case of syringomyelia in which separate cavities occurred in the cervico-dorsal and lumbo-sacral regions. A tumour was associated with the upper cavity. This tumour contained cells similar to those found in the walls of the cavity. Tamaki and Lubin[7] presented a case in which the transition from ependymal inclusions to cavity formations was clearly shown. Linear arrangement of ependymal cells was present throughout the whole cord, partially lining cavities in some segments, and existing purely as rests in other segments where there was no cavitation.

The series of cases of "familial lumbo-sacral syringomyelia" presented in this paper demonstrate the diffuse-

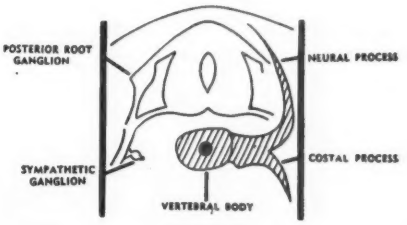

FIGURE VIII.

ness of the myelodysplastic process. In the lumbo-sacral region there is evidence of gliosis and possibly cavitation.

There is also pathological change in the cervico-dorsal region in most of these cases, as evidenced by the sensory disturbances in the hands. That these changes are very slowly progressive is seen by observation of the patient in Case I, in which sensory changes in the hands appeared sixteen years after the onset of symptoms of the disease. The cervico-dorsal lesion is one of gliosis, or cavitation, and affects the central region of the cord in at least some of the cases, producing dissociation of anæsthesia.

The exact manner in which this "primary ependymal gliosis" can cause trophic disturbances of the limbs is uncertain, but it is presumed that the cause is primarily an autonomic nervous disturbance, resulting in vasomotor and neurotrophic phenomena. As regards the bony changes in the feet, the classic work of Leriche and Policard has

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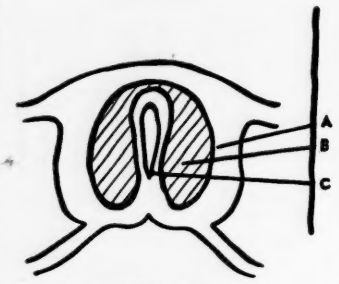
shown that osseous rarefaction is due to an increase in local circulation. This is presumably the causal factor in the present cases.

The ætiology of intramedullary tumours and the relation between syringomyelia and tumours are not yet established, but it would seem that some intramedullary tumours, particularly ependymomas and probably dermoid tumours, are a variant of myelodysplasia. The difference between these tumours and ependymal rests is probably merely a matter of rate of growth.

In the light of these observations, syringomyelia can be considered as a "heterotopic and benign process which may be differentiated histologically from tumour formations by lack of characteristic proliferation and invasive gliosis".[8]

Study of the pathogenetic factors provides the answer to the question: "Why does this congenital condition so often become manifest late in life?"

The primary ependymal glia may give rise to symptoms only if some secondary change, such as proliferation, cavitation, gliosis or hæmorrhage, occurs. This secondary change may occur at any time during life, or not at all. Symptoms in many cases commence with trauma of the lower limbs. This illustrates the fact that trauma to a poorly innervated extremity may bring to light symptoms of a latent nervous disease.



A. MARGINAL ZONE
B. MANTLE ZONE
C. EPENDYMAL ZONE

FIGURE IX.

The Inherited Constitution, or "Status Dysraphicus".

It has been found that examination of the family of a patient affected with syringomyelia reveals other disturbances of a very varied character.

Fuchs,[1] in 1903, described the familial association of trophic and vasomotor changes in the lower limbs with *spina bifida occulta*, sensory disturbances, and other defects.

From this, and similar observations, the concept of "*status dysraphicus*" arose.

This term was first used by Bremer[9] in 1926 to designate the constitutional disorder which appears to be an underlying factor in the ætiology of syringomyelia and other forms of myelodysplasia.

Bremer's work consisted in the careful observation of the apparently healthy members of the families of patients suffering from syringomyelia. He found in these people a striking incidence of certain disorders, of which he noted in particular: (i) abnormalities of the spinal column, particularly *spina bifida occulta* and kyphoscoliosis, (ii) trophic and vasomotor changes in the lower limbs, (iii) acrocyanosis, particularly of the fingers, (iv) weakness of the sphincters, (v) disturbances of sensation in the legs, sometimes of dissociated type, (vi) deformities of the feet, particularly club-foot and *pes cavus*, (vii) skin changes, such as *fovea coccygea* and nævus of the lumbar area, (viii) sternal abnormalities, particularly funnel chest, (ix) inequality of the mammary glands, and (x) greater span than height.

These disorders are the most common manifestations of the dysraphic state, and have at times, been described as *formes frustes* of syringomyelia.

The existence of *status dysraphicus* has long been disputed, but it has been proved most convincingly by recent work of Curtius,[10] Bremer,[11] Curtius and Lorenz,[12] Mulvey and Riely,[13] Bogaert[14] and others.

The name *status dysraphicus* indicates the constitutional type of condition associated with spinal dysraphism, that is, with faulty formation of the spinal column and abnormal closure of the cord. This abnormality may find partial expression in the varied local anomalies and clinical manifestations mentioned above.

The somewhat confusing terminology may be clarified by the following scheme:

Curtius and Lorenz,[12] after extensive work, concluded that *status dysraphicus* was a definite clinical and developmental type of condition. In 35 cases of *status dysraphicus* in which they made observations the most obvious signs were enuresis in fourteen cases, funnel chest in eleven cases, *pes cavus* in four cases, mammary abnormalities in two cases, and kyphoscoliosis in one case.

These authors, from observations in a series of 6868 cases, in eight different surveys showed that the incidence of *spina bifida occulta* in normal people is approximately 17·3%. The incidence of *spina bifida occulta* in 329 cases of enuresis was found to be 59·5%. From these figures they concluded that: (i) 17·3% of people are dysraphic; (ii) the theory of the functional or psychogenic origin of enuresis

Status Dysraphicus (an inherited constitutional defect)
Spinal Dysraphism
Associated Defects
Neural Defect Myelodysplasia including Syringomyelia
Bony Defect Spina Bifida Kyphoscoliosis et ceteras

FIGURE X.

is wrong, as in a large proportion of cases the enuresis is a sign of *status dysraphicus*.

It must be realized that, although 17·3% of people are dysraphic, neurological signs develop only in a small proportion, and that when the dysraphic process does give rise to clinical pictures, they may be confused and varied. Thus, in a dysraphic family, one member may present himself with acrocyanosis and another with enuresis.

In the series personally observed and presented in this paper, it was unfortunately possible to examine only one of the healthy members of the family. This patient, a man, aged forty-three years, showed obvious signs of *status dysraphicus*: (i) slight kyphoscoliosis; (ii) cold feet, which were very difficult to keep warm, and which sweated profusely even when cold; (iii) very thick callosities on the soles of the feet; (iv) very slow healing of any minor abrasion of the feet; (v) a well-marked *spina bifida occulta* of the first sacral segment.

Curtius[10] considers that the dysraphic state arises as a primary disturbance of the *Anlage*, resulting from an abnormal mutation of the main gene controlling the normal development of the medullary tube. As this is one of the earliest embryological processes, it can be seen that its disturbance may produce pleomorphic effects.

The neural effects may be the result either of true myelodysplastic changes in the cord or of an inherited constitutional biological inferiority affecting particularly the nervous system.

Recently the subject of inherited abnormal constitutions has been fully discussed by Bauer.[15]

Conclusions.

Errors of development of the spinal cord and column are by no means uncommon; about 17% of the population show radiological evidence of *spina bifida occulta*. This is the mildest form of the disorder, or "*status dysraphicus*".

The more severe forms of dysraphism may show many and varied clinical manifestations.

Evidence is accumulating which suggests that many apparently unrelated disorders, such as primary cervical syringomyelia and acrocyanosis, may indeed be end results of a common disturbance of development.

Summary.

1. A new concept is put forward regarding the significance of developmental errors of the spinal cord and column.
2. The natural history is discussed of a rare disease which is one of the manifestations of abnormal spinal cord development.
3. A discussion is presented of the nature of this disease and its relation to the general problem of anomalous spinal cord and column development.
4. A discussion is presented of the significance of the anomalous development, or "spinal dysraphism", and its possible clinical manifestations.

Acknowledgement.

I am indebted to Mr. Woodward-Smith, of the medical artistry department of the University of Sydney, and to the staffs of the pathology and radiology departments of the Royal Prince Alfred Hospital.

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THE POST-MORTEM BLOOD SUGAR.

By J. I. TONGE and J. S. WANNAN,
Laboratory of Microbiology and Pathology, Brisbane.

NOT infrequently pathologists, and particularly those conducting coronial autopsies, are confronted with cases of sudden death in which clinical data are either inadequate, misleading or completely absent. Among the causes of sudden death, diabetic and hypoglycæmic coma must be considered, and their diagnosis may easily be missed as the autopsy findings are likely to be insignificant.

The most reliable histological finding in cases of diabetes is the so-called intercapillary glomerulosclerosis, the peculiar spherical hyaline masses in the central portions of the glomerular lobules, first described by Kimmelstiel and Wilson in 1936.(1) The incidence of this diagnostic feature has been noted by various authors.(2)(3)(4)(5) It is said to be absent in individuals under thirty years of age, and less than 10% of diabetics between thirty and fifty years show these glomerular changes. In persons over fifty years of age the lesions were found in only 22% of male and 36% of female diabetics.

With these facts in mind it was realized that an investigation of the post-mortem blood sugar content should be made to determine what significance could be attached to it and what modifying factors, after death, affect its interpretation. It was hoped that one could determine by post-mortem blood sugar estimations whether a subject had died from diabetic coma or coma due to insulin overdosage. Hill(6) has stated that significant quantities of residual glucose are present in the majority of cases of asphyxia, shock, acute coronary closure, increased intracranial pressure and fluoride poisoning, indicating that agonal hyperglycæmia was present. It was thus hoped, further, that the post-mortem blood sugar estimation would provide additional corroborative evidence in death attributable to asphyxia, cases which are of extreme medico-legal importance and often providing few signs at autopsy.

Collection of Samples.

Hamilton-Paterson and Johnson(7) have stated that the glucose content of the blood rises in some parts of the body and falls in others after death; it is thus of extreme importance that blood specimens for the estimation of glucose content should be taken from a site in which the blood sugar most closely approaches the agonal level.

After death glycogen disappears rapidly from the liver, with the formation of glucose, and this glucose diffuses through the inferior *vena cava* to the right side of the heart. Thus blood taken from the right side of the heart may present glucose levels far in excess of those found in blood from the left atrium or ventricle. In livers with depleted liver glycogen before death this diffusion of glucose does not occur. Hill showed by experiment that in the normal dog the sugar content of the blood in the right auricle rose rapidly after death to a level of 400 milligrammes *per centum* in two hours and to 720 milligrammes at the end of twenty-four hours. The sugar content of the blood from the left ventricle of the same animal fell from 93·5 milligrammes to 28·7 milligrammes *per centum* in five hours. Hill also showed that no post-mortem rise in the sugar content of the right atrium occurred in an animal which had fasted for three days prior to the experiment. He was further able to show that in normal dogs in which all vessels to and from the heart were doubly ligated, comparison of the blood sugar levels of the right atrium with that of the inferior *vena cava* indicated that the previously observed post-mortem rise in the right atrium came from the inferior *vena cava*.

Hill quotes Balachowsky and Ginsburg, who noted that a post-mortem rise in the blood sugar content of the right atrium failed to develop in hepatectomized animals, and they concluded that the liver was the source of the sugar causing this increase. Hill quotes Burghard and Paffrath, who observed that glycogenolysis occurred rapidly in isolated liver tissue and that there was no change in the total carbohydrate content of such tissue up to thirty-six hours. Simpson and Macleod(8) have observed in similar experiments that the increase in dextrose paralleled the disappearance of glycogen from the liver.

It might be considered that the rise in the blood sugar content after death in the right atrium could be due to breakdown of glycogen in the cardiac muscle. If this were the case, however, a greater increase would be expected in the left ventricle than in the right, owing to the thicker muscle, but, as our results will show, this does not occur.

The increase in the sugar content of the blood in the right atrium and inferior *vena cava* would thus appear to be due to diffusion from the liver through the hepatic veins. The left side of the heart is apparently not affected, as the lungs act as an efficient barrier to this diffusion along the blood vessels.

Early in our investigations we were not aware of this difference in the blood sugar levels between the two sides of the heart and took samples of blood from the right atrium only. It was thus that our first observations on 63 blood specimens, taken from autopsies at random, gave consistently high figures—for example, 55% over 200 milligrammes *per centum* of reducing substance. It was realized

that these figures were obviously fallacious. Schmidt and Carey,[9] using blood obtained by cardiac puncture, published their results of a series of 33 cases and their findings are of little value, as no indication is given whether arterial, venous or mixed blood was used for the sugar determination.

Presence of Reducing Substances other than Glucose in Post-Mortem Blood.

In normal blood sugar estimations not all the material determined by the usual reduction methods is glucose. Hiller, Linder and Van Slyke[10] found in blood 15 to 30 milligrammes *per centum* of reducing substances that could not be removed by either spontaneous glycolysis or yeast fermentation. These non-glucose reducing substances are found chiefly in the blood corpuscles according to Somogyi,[11] and he believes that they are mainly composed of thioneine and glutathione. By modern methods of precipitation and highly specific reduction procedures the effect of these "other reducing substances" on the normal

Table I.

Showing Comparison of Glucose Levels in Post-mortem Blood Obtained by Hagedorn-Jensen Method with "Glucose" Level Obtained by Schaffer-Hartmann Technique.

Case.	Site of Blood Sampling.	Hagedorn-Jensen Total Reducing Substances.[1]	Hagedorn-Jensen "Other Reducing Substances".[1]	Hagedorn-Jensen True Glucose.[1]	Schaffer-Hartmann "Glucose".[1]
3799	Left ventricle ..	145	54	91	116
	Right atrium ..	213	83	130	180
3800	Left ventricle ..	195	100	95	124
	Right atrium ..	491	239	292	356
3801	Left ventricle ..	286	159	127	191
	Right atrium ..	270	130	140	168
3802	Left ventricle ..	188	95	93	104
	Right atrium ..	370	170	200	249
3803	Left ventricle ..	61	56	5	11
	Right atrium ..	66	60	6	11
3903	Left ventricle ..	331	161	170	197
	Right atrium ..	495	236	259	268
3905	Left ventricle ..	343	227	116	185
	Right atrium ..	562	478	84	370
3908	Left ventricle ..	70	57	13	35
	Right atrium ..	24	26	Nil	23
3909	Left ventricle ..	310	191	119	150
	Right atrium ..	195	163	32	58
3913	Left ventricle ..	270	89	181	203
	Right atrium ..	551	252	299	446
B1	Left ventricle ..	113	71	42	58
	Right atrium ..	202	120	82	122
3917	Left ventricle ..	224	111	113	133
	Right atrium ..	416	225	191	143

[1] These results are expressed in milligrammes per 100 millilitres of blood.

blood sugar determinations has been reduced to a minimum. Van Slyke[12] gives the figure as not more than 10 milligrammes *per centum*. These "other reducing substances" in the living subject are not affected by physiological or pathological conditions that greatly alter the concentration of glucose in the blood.

Harrison[13] stresses, however, that it is not correct to calculate true glucose by deducting non-fermentable reducing substance, say 10 milligrammes, from the "enhanced glucose" value, because varying proportions of non-fermentable reducing substances are included according to the sensitivity of the method of blood sugar determination employed.

Since these reducing substances are present in normal blood from the living subject it was considered that they might be partly responsible for the high figures we were obtaining in post-mortem blood, since blood taken at autopsy probably contained considerable amounts of these "other reducing substances" as a result of autolysis after death.

To determine the amounts of these "other reducing substances", the total reducing substances were estimated and then, after yeast fermentation to remove the glucose, a further estimation was carried out. In this way the total reducing substances (T.R.S.), true glucose, and "other reducing substances" (O.R.S.) were determined. We employed throughout the Hagedorn-Jensen method of blood sugar estimation.

Both the Hagedorn-Jensen and the Harding modification of the Schaffer-Hartmann technique, as outlined by King,[14] were designed for determining glucose in normal blood, which, as stated above, is low in these "other reducing substances". It was found, however, in post-mortem blood that the Hagedorn-Jensen method was very sensitive to these "other reducing substances" and that the figures for the total reducing substances were always considerably higher when that method was used than when the Schaffer-Hartmann technique was employed.

As other workers,[6][7] using the copper sulphate methods of reduction, have reported results, it was decided to run in parallel with the ferricyanide (Hagedorn-Jensen) method a series in which the Schaffer-Hartmann technique was used. This was done to determine to what extent the Hagedorn-Jensen technique was affected by these "other reducing substances". The total reducing substances, "other reducing substances", and true glucose levels obtained, were compared with the "glucose" level obtained by the Schaffer-Hartmann technique without fermentation. The results are set out in Table I.

Table I shows the greater susceptibility to reduction of the ferricyanide (Hagedorn-Jensen) by the "other reducing substances" as compared with their effect on copper sulphate (Schaffer-Hartmann). This difference has previously been noted by Munday and Seibert[15] in their paper on the determination of polysaccharide in tuberculin. If in a graph the Schaffer-Hartmann "glucose" value is plotted against the true glucose value as estimated by the Hagedorn-Jensen technique, a straight-line relationship exists.

As a result of these findings it was noted that post-mortem blood contains considerable quantities of non-fermentable reducing substances, products of autolysis,

Table II.

Tube.	Estimation.	Water.	Yeast.	Blood.	Protein Precipitant.
	Total reducing substances—				
1	Left ventricle	3·3	—	0·2	0·5
2	Right atrium	3·3	—	0·2	0·5
	Other reducing substances—				
3	Left ventricle	2·3	1·0	0·2	0·5
4	Right atrium	2·3	1·0	0·2	0·5
5	Yeast blank	2·5	1·0	—	0·5
6	Protein precipitant blank ..	3·5	—	—	0·5

which render both the Hagedorn-Jensen and Schaffer-Hartmann methods of estimating glucose erroneous, and that the true glucose level can be determined only after yeast fermentation. It was also seen that blood taken from the right side of the heart gave figures for true glucose higher in almost every case than those obtained with blood taken from the left side, owing to post-mortem glycogenolysis and diffusion of glucose through the inferior *vena cava*. It is thus essential that blood be obtained at post-mortem examination from the left ventricle, as this gives the closest approximation to the true blood glucose level existing at the time of death.

Method of Determining the "True Glucose" Level.

Difficulty was experienced at first in minor points of technique in carrying out the yeast fermentation, and for this reason the method we employed and found satisfactory is set out in some detail.

True glucose can be determined only by specific fermentation with live yeast. The absolute specificity and wide range of pH (5·0 to 9·0) of this reaction make it quite applicable to post-mortem blood, and the true glucose content may be estimated by the quantitative difference in reduction value before and after yeast fermentation. The first reduction gives a quantitative estimate of the total reducing substances and the second reduction value obtained after yeast fermentation gives the non-fermentable

TABLE III.

Groups.	Number of Cases.	Average True Glucose.			Significance.
		Right Atrium.	Left Ventricle.	Difference Between (R–L).	
Control groups—					
Natural causes	22	101·9±22·98	67·7±10·12	34·2±18·04	N.S.[1]
Sudden death	25	126·4±28·89	58·2±11·18	68·1±23·23	H.S.[3]
Miscellaneous	10	57·3±26·61	33·2±12·35	24·1±17·46	N.S.
Test groups—					
Asphyxia	23	157·0±18·47	113·4±13·80	43·7±18·37	S.[2]
Coronary disease	34	181·2±16·58	93·1± 9·86	88·1±14·89	H.S.
Raised intracranial pressure	16	124·8±22·84	100·8±17·90	23·9±14·79	N.S.
Diabetes	3	426·0±98·08	399·7±100·60	26·3± 8·57	N.S.
Chronic alcoholism (liver damage)	10	29·5± 8·06	32·7± 8·73	−3·2± 8·22	N.S.

[1] N.S. = not significantly different from zero at the 5% level.
[2] S. = significantly different from zero at the 5% level.
[3] H.S. = significantly different from zero at the 1% level.

reducing substances or "other reducing substances"—the difference being due to true glucose.

Inconsistent results were obtained through the prolonged action of the yeast, which itself produces reducing substances, so optimal conditions were obtained by using a concentrated well-washed yeast suspension acting over a short period of time.

Commercial brands of compressed yeast were found to be unsuitable even after extensive washing, owing to the large blank reducing values manifested by traces of sugars (and other carbohydrate material of high reducing value) still present. A culture of pure baker's yeast constantly maintained in liquid wort was found quite suitable.

This fresh culture was subjected to at least six washings with distilled water and finally suspended in isotonic sodium sulphate solution (four volumes to each volume of centrifuged yeast cells). One millilitre of this suspension was found to be quite adequate for fermentation of all the glucose occurring in post-mortem blood and yet possessed, *per se*, quite an insignificant reducing value. This concentrated suspension was kept in the refrigerator and a blank titration value was determined each day before use. If the blank titration value began to show an appreciable increase, the yeast was rewashed and resuspended in isotonic sodium sulphate solution as before.

The yeast suspension was allowed to act at room temperature for fifteen minutes, the tubes were then centrifuged, two millilitres of the supernatant fluid were added to two millilitres of ferricyanide solution, and the reducing value was determined by the Hagedorn-Jensen method.

The tube "set-up" for each post-mortem blood was as shown in Table II.

Glycolysis.

It has been known since the time of Claude Bernard that sugar gradually disappears from blood when the blood is allowed to stand.[13] This glycolysis is brought about by an intracellular enzyme. Tolstoi[16] and others have stated that at body temperature from 10 to 20 milligrammes of glucose per 100 millilitres of blood per hour are broken down, the rate being apparently independent of the initial concentration of glucose in the blood. Recent workers are of the opinion that there is no difference between the glycolytic powers of normal blood and diabetic blood *in vitro*.[13] Glycolysis is retarded by chilling, accelerated by warming to body temperature, and ceases if the blood is heated to 58° C. Glycolysis is retarded by oxalate, checked by fluoride and markedly retarded by laking of the blood.[17] It proceeds more rapidly at alkaline reactions, reaching a maximum at a pH of 8 to 9,[18] and its rate can be related quantitatively to the number of leucocytes. It can thus be seen that *in vitro* glycolysis is influenced by many factors and still more complex does the subject become when attempts are made to determine the rate of glycolysis *post mortem*.

It is clear that the complex changes taking place in the body after death make an accurate study of the rate of glycolysis within the cadaver impossible. Hamilton-Paterson and Johnson found that glycolysis proceeds very rapidly in post-mortem blood from non-diabetic subjects in both the blood and cerebro-spinal fluid. In twenty-four of their cases maximum glycolysis had occurred when the first estimation was made (seven to sixteen hours after death). They observed, however, that in patients who had had hyperglycæmia no appreciable glycolysis was noted even up to fifty-four hours after death. They also found that glycolysis was similarly retarded in the cerebro-spinal fluid of people dying with hyperglycæmia. On the other hand, in subjects with a normal blood sugar content at the time of death they found in their series that glycolysis proceeded rapidly and was complete in from three and a half to seven hours. In-vitro experiments show no such difference between hyperglycæmic and normal blood.

Hamilton-Paterson and Johnson further state that refrigeration makes little difference to the rapidity of glycolysis, as the process was in normal cases almost complete before the internal organs were cooled. They observed that six to seven hours elapsed before the temperature of the internal organs was appreciably lowered in bodies refrigerated shortly after death. They found that during the period at which the internal organs were

TABLE IV.
Regression Coefficients.

Groups.	Minimum Time Since Death in Hours.	All Times.				Up to Eight Hours since Death.			
		Number of Cases.	Average True Glucose.			Number of Cases.	Average True Glucose.		
			Right Atrium.	Left Ventricle.	(R–L).		Right Atrium.	Left Ventricle.	(R–L).
Natural causes	2	21	2·83±1·60	1·21±0·66	1·62±1·31	9	15·36± 9·20	8·90± 5·24	6·46± 5·48
Sudden death	2	24	1·86±1·82	−0·65±0·71	2·51±1·40	5	6·77±17·02	−5·15±10·99	11·92± 6·96
Miscellaneous	7	10	−1·16±1·14	−0·75±0·50	−0·41±0·78				
Asphyxia	6	23	−0·45±1·49	0·67±1·11	−1·12±1·47	6	−28·75±16·67	−6·0±27·42	−22·75±34·48
Coronary disease	1½	34	−0·04±1·29	−0·01±0·77	−0·03±1·16	11	−10·12±12·78	−17·33±5·96	7·21±13·05
Raised intracranial pressure	2½	16	−2·14±4·56	0·54±3·60	−2·68±2·89	4	−8·35±23·37	4·25±18·20	−12·6 ± 6·33
Chronic alcoholics	6	10	−0·52±0·63	−1·17±0·58	0·65±0·63				

TABLE V.
Cases with Death Due to Natural Causes.

Case.	Hours P.M.	Right Atrium.			Left Ventricle.			Diagnosis.
		T.R.S.[1]	O.R.S.[2]	True Glucose.	T.R.S.[1]	O.R.S.[2]	True Glucose.	
3989	2	97	70	27	150	102	48	Congestive cardiac failure.
3850	3	106	82	24	84	71	13	Congestive cardiac failure.
3830	3½	159	100	59	206	170	36	Myocardial degeneration.
3871	4	45	31	14	61	27	34	Asthma; congestive cardiac failure.
3806	6	96	47	49	114	56	58	Ruptured aortic aneurysm.
3899	6	159	96	63	168	99	69	Pulmonary œdema; congestive cardiac failure.
3804	6½	39	25	14	61	50	11	Emaciation; pulmonary fibrosis.
3763	7½	168	98	70	150	90	60	Pneumonia.
3917	7½	416	225	191	224	111	113	Myocardial degeneration.
3849	10	88	73	15	70	68	2	Metastatic carcinoma of prostate.
3788	10	251	65	186	236	65	171	Congenital heart.
3811	11	127	86	41	124	128	Nil	Congenital heart.
3807	11½	197	92	105	99	50	49	Myocardial degeneration.
3844	19	298	170	128	262	152	110	Hypertension; cardiac failure.
3824	19	370	213	157	97	52	45	Hypertension; myocardial degeneration.
3939	23	184	132	52	184	134	50	Myocardial degeneration.
3769	24	213	64	149	159	68	91	Arteriosclerosis; myocardial degeneration.
3852	24½	168	79	89	221	110	111	Hypertension; hæmorrhage.
3823	35	553	446	107	131	86	45	Nephritis.
3781	39	894	382	512	232	93	139	Myocardial degeneration.
3846	60½	70	32	38	138	43	95	Pneumonia.
3946	?	405	253	152	290	151	139	Pulmonary œdema; acute alcoholism.

[1] T.R.S. = total reducing substances.
[2] O.R.S. = "other reducing substances".
The figures represent milligrammes per hundred millilitres of blood.

at approximately 37° C., glycolysis destroyed 12·8 milligrammes of dextrose per hour.

Hill is of the opinion that intravascular and in-vitro glycolysis occur at approximately the same rate and are not influenced by clotting. Hamilton-Paterson and Johnson state, in summarizing the position, that post-mortem glycolysis occurs at a slower rate in the cadaver than *in vitro*. Little work has been done on the rate of glycolysis *post mortem* and quite obviously the subject is a complicated one. Our findings certainly suggest that there is an appreciable difference in the rate of glycolysis *in vivo* and *in vitro*. It will be seen from the following tables that even in the control cases (Tables V, VI and VII), in which it can be assumed that the agonal blood sugar content was normal, appreciable levels of glucose were detected in some, even as long as sixty hours after death.

Whilst it is undoubtedly important to note the time since death when collecting a blood specimen, one should refrain from jumping to the conclusion that hyperglycæmia was present at the time of death just because, for example, 144 milligrammes of glucose *per centum* are detected twenty-four hours after death. It will be shown that true glucose blood levels from the left ventricle are of little significance if under 200 milligrammes *per centum*. With levels of 200 or above, hyperglycæmia can be diagnosed.

TABLE VI.
Cases in which Death was Due to Severe or Violent Trauma.

Case.	Hours P.M.	Right Atrium.			Left Ventricle.			Diagnosis.
		T.R.S.[1]	O.R.S.[2]	True Glucose.	T.R.S.[1]	O.R.S.[2]	True Glucose.	
3832	2	241	132	109	224	125	99	Gunshot wound to head.
3933	3	530	474	56	462	375	87	Shock. Sudden blow on abdomen.
3795	5½	63	36	27	52	36	16	Multiple injuries. Rupture of liver. Hæmorrhage.
3860	8	414	190	224	255	129	126	Shock. Multiple fractures. Acute alcoholism.
3879	8	213	192	21	57	63	Nil	Head injury. Railway accident.
3874	9	59	45	14	25	27	Nil	Gunshot wound to brain.
3875	10	70	56	14	41	31	10	Gunshot wound to brain.
3847	10	177	118	59	117	83	34	Gunshot wound to head.
3805	10½	747	152	595	375	154	221	Electrocution.
3778	11½	222	54	168	195	68	127	Multiple injuries. Extradural hæmorrhage. Truck accident.
3826	11¾	88	86	2	77	88	Nil	Fractured skull. Multiple fractures.
3783	13	75	32	43	117	61	56	Multiple fractures. Hæmorrhage.
3800	13	491	239	252	195	100	95	Electrocution.
3808	15½	476	321	155	102	54	48	Crushed chest. Ruptured aorta.
3776	18	177	53	124	141	57	84	Fractured skull. Maceration of brain.
3867	20	191	149	42	52	60	Nil	Sudden incineration.
3887	23	213	156	57	186	95	91	Crush injury to chest. Railway accident.
3888	24	981	846	135	808	664	144	Scalding. Laceration of liver. Railway accident.
3865	26	621	287	334	110	85	25	Ruptured heart and liver. Multiple fractures.
3893	37	1700	1720	Nil	148	124	24	Fractured skull. Brain injury.
3969	38	213	188	35	88	80	8	Crush injury to chest.
3780	40	594	314	280	77	47	30	Fractured skull. Injury to brain.
3877	43	70	56	14	41	31	10	Head injury. Truck accident.
3765	75	740	385	355	132	72	60	Gunshot wound to head.
3822	?	141	97	44	179	118	61	Hæmorrhage. Cut throat.

[1] T.R.S. = total reducing substances.
[2] O.R.S. = "other reducing substances".
The figures represent milligrammes per hundred millilitres of blood.

TABLE VII.
Cases in which Death was Due to Miscellaneous Causes.

Case.	Hours P.M.	Right Atrium.			Left Ventricle.			Diagnosis.
		T.R.S.[1]	O.R.S.[2]	True Glucose.	T.R.S.[1]	O.R.S.[2]	True Glucose.	
3771	7	124	28	96	124	25	99	Anæsthesia. Pneumonia.
3908	11	24	26	Nil	70	57	13	Septic abortion. Septicæmia.
3796	13½	251	170	81	243	208	35	Tetanus.
3821	15	70	47	23	70	47	23	Abortion.
4059	16	708	437	271	290	193	97	Anæsthesia. Oxycephalus.
3787	20	131	50	81	110	50	60	Anæsthesia.
3803	20	66	60	6	61	56	5	Concussion.
4052	21	65	50	15	41	45	Nil	Anæsthesia.
3862	45	222	230	Nil	113	120	Nil	Lysol poisoning.
3840	89	2695	2700	Nil	2695	2700	Nil	Advanced putrefaction.

[1] T.R.S. = total reducing substances.
[2] O.R.S. = "other reducing substances".
The figures represent milligrammes per hundred millilitres of blood.

Results.

The total reducing substances, true glucose and "other reducing substances" were estimated in 145 samples of blood taken from the right atrium and left ventricle *post mortem*. Since Hill has stated that hyperglycæmia and glycosuria were often observed during life in asphyxia, in acute coronary occlusion and in cases of increased intracranial pressure we grouped our cases under these headings. Included also are three groups comprising cases of sudden death, death due to natural causes and a miscellaneous group to serve as controls. Our diabetics were grouped together and we also made a final group in which there was microscopic evidence of severe liver damage. Details of the individual groups are set out in Tables V to XII.

A statistical analysis of the mean true glucose levels from the right atrium and left ventricle and of the difference between these two figures is given below in Table III.

Chronic alcoholism is a group in which no differentiation between right and left is to be expected. The other groups all show that the average true glucose level in the blood from the right atrium is higher than that from the left ventricle. In three of the groups this difference is significant, while in the case of the "natural causes" group the difference is approaching significance at the 5% level. Except in cases of chronic alcoholism, there is evidence that the average true glucose level in the blood from the right atrium is higher than in that from the left ventricle.

In addition, the standard errors of the determinations from the right atrium are higher than those from the left ventricle, except in the case of the diabetic and chronic alcoholism groups. The standard error indicates the degree of dispersion or range of values, and since the determinations from the left ventricle have the smaller standard error, it is preferable to use these.

The increase, on the average, of the true glucose determinations from the right atrium over those from the left ventricle and the greater disperson of the former could both be caused by diffusion of glucose to the right atrium. If diffusion were the cause, the values for the right atrium should show some trend with time.

To determine whether the data showed any trend with time, an equation of the type $g = a + bt$, where g is the glucose and t the time in hours, was fitted. First, all times were considered and then only those cases up to eight hours since death. The regression coefficients, b, together with their standard errors, are given in Table IV. Only one isolated value is significantly different from zero—that is, −17·33 for coronary disease in the "up to eight hours" group. Thus the data contain no evidence of any significant trend with time.

The groups "Natural causes", "Sudden death" and "Coronary disease" show positive values for the regression

TABLE VIII.
Cases in which Death was Due to Asphyxia.

Case.	Hours P.M.	Right Atrium.			Left Ventricle.			Diagnosis.
		T.R.S.[1]	O.R.S.[2]	True Glucose.	T.R.S.[1]	O.R.S.[2]	True Glucose.	
3802	6	370	170	200	188	95	93	Drowning.
3825	6	571	439	132	152	79	73	Hanging.
3878	7	280	170	110	195	188	7	Compression asphyxia.
3854	7½	186	77	109	232	112	120	Drowning.
3818	8	166	81	85	262	140	122	Hang ng.
4164	8	213	81	132	200	180	20	Hang ng.
3810	9½	502	187	315	345	134	211	Hang ng.
3838	? 12	280	69	211	274	87	187	Carbon monoxide poisoning.
3794	12	106	59	47	52	41	11	*Status asthmaticus.*
3814	14	232	109	123	232	65	167	Suffocation.
3959	16	213	130	83	251	140	111	Hanging.
3773	16	213	128	85	191	110	81	Carbon monoxide poisoning.
3868	18	455	170	285	430	147	283	Hanging.
3774	19	446	215	231	159	121	38	Drowning.
3934	19	533	476	63	423	269	154	Drowning.
3848	20½	270	146	124	255	117	138	Choking.
3767	21	412	188	294	259	93	166	Drowning.
3775	24	302	124	178	141	85	56	Hanging.
3833	27½	473	177	296	148	70	78	Carbon monoxide poisoning.
3809	35	466	187	279	290	172	118	Hanging.
3905	43	562	478	84	343	227	116	Carbon monoxide poisoning. Incineration.
3909	45	195	163	32	310	191	119	Drowning.
3901	50+	259	145	114	298	159	139	Drowning.

[1] T.R.S. = total reducing substances.
[2] O.R.S. = "other reducing substances".
The figures represent milligrammes per hundred millilitres of blood.

TABLE IX.

Cases in which Death Resulted from Coronary Disease.

Case.	Hours P.M.	Right Atrium.			Left Ventricle.			Diagnosis.
		T.R.S.[1]	O.R.S.[2]	True Glucose.	T.R.S.[1]	O.R.S.[2]	True Glucose.	
3913	1½	551	.252	299	270	89	181	Coronary atherosclerosis. Myocardial degeneration.
3761	3	370	125	245	331	132	199	Coronary atherosclerosis. Myocardial degeneration.
3992	3	491	250	241	385	225	160	Coronary atherosclerosis. Myocardial degeneration. Acute alcoholism.
3799	3½	213	83	130	145	54	91	Coronary occlusion.
3792	4	290	125	165	88	50	38	Coronary atherosclerosis. Hypertension.
3813	4	392	183	209	168	72	96	Coronary insufficiency.
3791	5½	306	85	221	199	85	114	Coronary insufficiency.
3777	6	691	296	395	102	46	56	Coronary occlusion.
3836	7	379	127	252	148	65	83	Coronary occlusion.
3762	7	168	59	109	127	59	68	Coronary insufficiency.
3855	8	241	125	116	88	29	59	Coronary occlusion.
3841	11	159	90	69	159	80	79	Coronary insufficiency. Hypertension.
3835	11½	213	131	82	77	21	56	Coronary insufficiency. Silicosis.
3798	12	395	137	258	97	43	54	Coronary occlusion.
3827	12½	370	220	150	240	143	97	Coronary insufficiency.
3851	14½	290	184	106	124	32	92	Coronary occlusion.
3856	14½	177	79	98	110	54	56	Coronary occlusion.
3898	15½	217	103	114	355	160	195	Coronary thrombosis.
3834	16	240	136	104	70	36	34	Coronary insufficiency.
3873	17	310	102	208	141	56	85	Coronary thrombosis.
3837	17½	385	124	261	88	60	28	Coronary atherosclerosis.
3812	17½	70	40	30	41	18	23	Coronary insufficiency.
3760	18	177	74	103	110	51	59	Coronary occlusion.
3801	19	270	130	140	286	159	127	Coronary atherosclerosis. Myocardial degeneration.
3820	19	388	251	137	59	61	Nil	Coronary occlusion.
3870	20	155	45	110	131	58	73	Coronary atherosclerosis. Aortic stenosis.
3766	22	184	72	112	110	76	34	Coronary atherosclerosis. Hypertension.
3872	22	419	138	281	336	127	209	Coronary thrombosis.
3864	23½	679	267	412	392	160	232	Coronary occlusion.
3772	24	430	54	376	177	73	102	Coronary occlusion.
3793	28	195	83	112	155	61	94	Coronary occlusion.
3843	41	587	439	148	146	98	48	Coronary occlusion.
3845	41	598	483	115	247	120	127	Coronary insufficiency.
3797	67	530	267	263	232	117	115	Coronary occlusion.

[1] T.R.S. = total reducing substances.
[2] O.R.S. = "other reducing substances".
The figures represent milligrammes per 100 millilitres of blood.

coefficients of the differences between the right atrium and the left ventricle on time (cases up to eight hours). It is to be noted that these groups contain more cases in which examinations were made at shorter intervals after death. The "Asphyxia" group, which shows a large negative coefficient, contained no cases with the death to post-mortem examination interval less than six hours, whilst the "Raised intracranial pressure" group contained only one case examined at two and a half hours, the remaining cases being at four, six and eight hours.

Although the number of cases concerned is too small to allow any definite conclusions to be drawn, the values in Table IV are consistent with the hypothesis that diffusion of glucose to the right atrium from the liver takes place fairly rapidly (within a period of two to three hours after death).

The Control Cases.

Our control cases consisted of three groups, namely, cases in which death occurred from natural causes, cases in which patients died suddenly as a result of severe or violent trauma, and a miscellaneous group. Details of our findings in these three groups are set out in Tables V, VI and VII.

A perusal of these figures reveals how difficult is the interpretation of an isolated true glucose level from the left ventricle. Since it may be presumed that in these cases there was an agonal blood sugar content within normal limits, the wide variation of these true glucose levels at varying times since death makes one realize that any figure for the true glucose level from the left ventricle under 200 milligrammes *per centum* is probably of no significance. The tables also clearly demonstrate the misleading true glucose values which are obtained in blood from the right atrium, as well as the wide variation in the "other reducing substances" as determined by the Hagedorn-Jensen technique.

If one regards levels for the true glucose from the left ventricle as being of little significance below 200 milligrammes *per centum*, it will be seen that of the fifty-seven control cases only one, Case 3805 in Table VI, gave evidence of hyperglycæmia. The individual in this case was accidentally electrocuted and apparently died instantaneously.

Case 3840 in Table VII is of interest in that the body was examined eighty-nine hours after death, having been recovered from the river in an advanced state of putrefaction. As would be expected, no glucose is present in the blood of either the right or left side of the heart, but the enormous increase in the non-glucose reducing substances evenly distributed in the blood through both sides of the heart is consistent with advanced decomposition.

Cases of Asphyxia.

Included in the twenty-three cases of asphyxia examined are eight in which death was due to hanging, seven to drowning, four to carbon monoxide poisoning, one to *status asthmaticus*, one to suffocation, one to choking, and one due to compression asphyxia. The detailed findings are set out in Table VIII. It will be seen that in two cases of hanging there are true glucose levels from the left ventricle above 200 milligrammes *per centum*, and these almost certainly represent agonal hyperglycæmia.

Hill, in his series of eight cases of asphyxial death, found hyperglycæmia in only one, a figure of 608 milligrammes *per centum* of true glucose from the left ventricle of a person who hanged himself; the blood was examined sixteen hours after death. For the remaining seven cases the true glucose levels varied from 98 milligrammes *per centum* to nil, the blood being examined between two and a half to twenty hours after death. Hill, however, states: "If allowance is made for the glycolysis which undoubtedly occurred before the internal organs had cooled, it becomes apparent that the agonal blood sugar in these cases must have exceeded normal limits." We, however, cannot accept

TABLE X.
Patients Dying with Raised Intracranial Pressure.

Case.	Hours P.M.	Right Atrium.			Left Ventricle.			Diagnosis.
		T.R.S.[1]	O.R.S.[2]	True Glucose.	T.R.S.[1]	O.R.S.[2]	True Glucose.	
3799	2½	345	83	262	232	72	160	Subdural and pontine hæmorrhage.
3962	4	119	71	48	184	164	20	Subdural and cerebral hæmorrhage.
3869	6	241	93	148	141	45	96	Cerebral hæmorrhage.
3770	8	232	59	173	213	50	163	Cerebral tumour.
3831	9	416	228	188	290	157	133	Ruptured cerebral aneurysm.
3952	10	70	53	17	186	124	62	Cerebral tumour.
3826	11½	88	86	2	77	88	Nil	Cerebral hæmorrhage. Fractured skull.
3768	13	145	36	109	120	45	75	Cerebellar hæmorrhage.
4004	13½	251	110	141	195	92	103	Cerebral hæmorrhage.
3859	14	409	183	226	124	58	66	Cerebral hæmorrhage.
3785	15	127	94	33	148	103	45	Subdural hæmorrhage.
3858	15	491	272	219	526	266	260	Cerebral hæmorrhage.
3861	16	106	76	30	177	104	73	Subdural hæmorrhage.
3790	17½	186	61	125	282	104	178	Cerebral hæmorrhage.
3903	20	495	236	259	331	161	170	Cerebral hæmorrhage.
3819	20	63	47	16	48	39	9	Extradural hæmorrhage.

[1] T.R.S. = total reducing substances.
[2] O.R.S. = "other reducing substances".
The figures represent milligrammes per hundred millilitres of blood.

this statement, as such wide variations exist in the rate of glycolysis *post mortem*, as evidenced by the control series, that it is not safe to calculate backwards and to presume hyperglycæmia as being present at the time of death when true glucose levels under 200 and certainly under 100 milligrammes are obtained, even many hours after death.

In many so-called asphyxial deaths and certainly in hanging, other factors, such as cerebral anæmia and nervous stimulation, play a part, and asphyxia is only one factor.[19] To us it would appear that examination of the post-mortem blood sugar is of little value in providing corroborative evidence of an asphyxial death. This is indeed unfortunate.

Cases in which Death was Attributable to Coronary Occlusion.

Included in this group are thirty-four cases in which death was attributed to blockage of the coronary arteries, either by a thrombus or atherosclerosis. Of the cases in this group, three showed a true glucose level from the blood of the left ventricle suggestive of agonal hyperglycæmia. Details of the findings for this group are set out in Table IX.

Cases in which Death was Accompanied by a Raised Intracranial Pressure.

In the group in which death was accompanied by raised intracranial pressure are fourteen cases of cerebral hæmorrhage, either extradural, subdural, subarachnoid, or hæmorrhage in the substance of the brain; also two patients with cerebral tumours. Each hæmorrhage and tumour was of sufficient size to have caused a pronounced rise in the intracranial pressure. A level for true glucose from blood in the left ventricle of the heart at a hyperglycæmic level was found in one case only (Case 3858). Details of the findings in this group are set out in Table X.

Glycosuria was present *post mortem* in four of the cases of cerebral hæmorrhage, and acetone was detected in the urine in one instance. In the case with evidence of hyperglycæmia there was a trace of reducing substances in the urine at post-mortem examination, but in the other three cases in which glycosuria was present there was no evidence of hyperglycæmia.

Diabetics.

Unfortunately only five diabetics are included in our series. Only three of these cases are included in the statistical survey (Table III), as in Case 4150 blood was taken from the aorta alone, and in Case BGH2 diabetes was not the primary cause of death. Four of the five diabetics died in coma; three a few hours after admission to hospital; the fourth in the street. The fifth diabetic (BGH2) died from congestive cardiac failure after five weeks in hospital.

True glucose levels in the blood from the left ventricle of three and from the aorta of the fourth patient dying in diabetic coma were definitely hyperglycæmic. The fifth case (BGH2), that of a controlled diabetic of long standing with post-mortem histological evidence of intercapillary glomerulosclerosis, was included in the diabetic group, but the patient died from an intercurrent disease. In this instance the blood sugar levels did not differ from those of the control series.

Glycosuria and ketonuria were present *post mortem* in two of the cases in which death occurred from coma, but unfortunately no urine was available for examination in the other three. Details of the findings in these five cases are set out in Table XI.

Although the examination of the urine may often provide the clue to the cause of death in diabetic coma, urine is

TABLE XI.
Cases in which Death was Due to Diabetes.

Case.	Hours P.M.	Right Atrium.			Left Ventricle.			Diagnosis.
		T.R.S.[1]	O.R.S.[2]	True Glucose.	T.R.S.[1]	O.R.S.[2]	True Glucose.	
BGH2	14	202	120	82	113	71	42	Congestive cardiac failure. Diabetes.
BGH1	23	847	316	531	795	274	521	Diabetic coma.
BGH3	17½	735	218	517	655	177	478	Diabetic coma.
4162	27	385	155	230	290	90	200	Diabetic coma.
4150[3]	35½	—	—	—	780	170	610	Diabetic coma.

[1] T.R.S. = total reducing substances.
[2] O.R.S. = "other reducing substances".
[3] The blood was collected from the ascending aorta and no specimen was obtained from the right atrium.
The figures represent milligrammes per hundred millilitres of blood.

TABLE XII.
Cases showing Liver Damage at Post-mortem Examination.

Case.	Hours P.M.	Right Atrium.			Left Ventricle.			Diagnosis.
		T.R.S.[1]	O.R.S.[2]	True Glucose.	T.R.S.[1]	O.R.S.[2]	True Glucose.	
3927	6	106	89	17	159	78	81	Cut throat. Chronic alcolohism.
3815	7	70	40	30	66	36	30	Hæmorrhage. Ruptured liver. Chronic alcoholism.
3966	12	260	204	56	331	280	51	Acute appendicitis. Peritonitis. Chronic alcoholism.
3941	17	52	46	6	48	50	Nil	Myocardial degeneration. Chronic alcoholism.
3829	21	146	93	53	117	86	31	Lysol poisoning. Chronic alcoholism.
3900	22	146	102	44	266	209	57	Head injury. Chronic alcoholism.
3863	26¼	152	80	72	131	80	51	Chronic alcoholism. Acute heart failure.
3995	31	99	98	1	195	196	Nil	Hæmorrhage. Duodenal ulcer. Chronic alcoholism.
3947	37	115	116	Nil	124	98	26	Chronic alcoholism. Myocardial degeneration.
3866	50+	181	165	16	132	138	Nil	Chronic alcoholism. Drowning.

[1] T.R.S. = total reducing substances.
[2] O.R.S. = "other reducing substances".
The figures represent milligrammes per hundred millilitres of blood.

not always available, and both glycosuria and ketonuria can occur in other conditions. From the results of this small series it will be seen that estimation of the true glucose level from the left ventricle will provide valuable evidence *post mortem* when diabetic coma is suspected.

Cases Showing Severe Liver Damage Post Mortem.

In our final group we collected ten cases, in each of which extensive liver damage was found microscopically. The liver damage was, in this group, due to chronic alcoholism and the cases were grouped together regardless of the primary cause of death. In each case histological examination revealed either extensive fatty change or cirrhosis with extensive atrophy of liver cells.

This grouping of cases was to determine whether there was a rise in the true glucose level of the right atrium compared with the true glucose level of the left ventricle as evidenced in the other groups. Perusal of Table XII reveals that there is a comparative rise in the true glucose level of the right atrium in only one case, and this only to a slight degree. The consistently low levels for the true glucose from the blood of the right atrium, as compared with all the other groups, is almost certainly due to the absence of liver glycogenolysis with diffusion of glucose, thus liberated, to the right side of the heart. This finding is consistent with the deficient glycogen storage of the damaged liver.

Conclusions and Summary.

To obtain as close an approximation as possible to the agonal blood sugar level, blood must be taken *post mortem* from the left side of the heart and the true glucose value estimated by yeast fermentation.

Blood estimations for true glucose in post-mortem blood taken from the right atrium or ventricle will be fallacious owing to liver glycogenolysis, occurring with a few hours of death, and diffusion of glucose to the right side of the heart via the hepatic veins and inferior *vena cava*. The lungs act as an efficient barrier, preventing this diffusion to the left atrium. Where severe liver damage is present with consequent ante-mortem glycogen depletion, this rise in the post-mortem true glucose blood level in the right side of the heart does not occur.

It is essential to employ yeast fermentation in obtaining the glucose level in post-mortem blood, owing to the presence of non-glucose reducing substances. Care must be taken in the preparation of the yeast for the fermentation test.

Glycolysis appears to occur at a slower rate in the cadaver than *in vitro*, and great caution should be exercised in the diagnosis of hyperglycæmia when a true glucose level in post-mortem blood is found to be raised even many hours after death. We regard any figure for true glucose from the blood of the left side of the heart as being of little or no significance below 200 milligrammes per 100 millilitres of blood.

With blood levels for true glucose in the left ventricle of 200 milligrammes per 100 millilitres or above, it is safe to say that hyperglycæmia existed at the time of death.

Hyperglycæmia, as a terminal event, can occur in conditions other than diabetes. A true glucose blood figure from the left ventricle at a hyperglycæmic level was found, apart from the four cases of diabetic coma, in six of our series of 145 cases. Of these six cases, two were asphyxial deaths due to hanging, two were cases of acute coronary occlusion, one was associated with cerebral hæmorrhage, and one with death due to electrocution.

Glycosuria, like hyperglycæmia, can be found in conditions other than diabetes, and was found at post-mortem examination in five cases of cerebral hæmorrhage and in one case of coronary occlusion, in none of which was there any suggestion of a diabetic history.

The post-mortem blood sugar level, provided due regard is paid to correct sampling and estimation, as well as caution in interpretation, can be of considerable value in the diagnosis of hyperglycæmia. Though no cases of hypoglycæmia were included in the series, it would appear that blood should be taken very soon after death for sugar estimations to be of any significance.

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CRIMINAL RESPONSIBILITY IN INCIPIENT PSYCHOSIS.[1]

By F. M. G. PRENDERGAST.
Perth.

As is well known, the McNaghten rules have been the criteria accepted by British law for more than one hundred years as to whether a person is responsible or not for his criminal actions. These rules stated very simply are that (*a*) if a person knows what he is doing and (*b*) if he knows that it is wrong, then he is held to be criminally responsible for his actions.

However, over the years it has been contended that these rules take too narrow a view of mental disorder. It has been pointed out, for instance, that they make no allowance for the melancholic who murders his children, knowing what he is doing and knowing that it is forbidden by the law, but yet driven on to the deed by an insane delusion that his children are suffering from an incurable disease. It has been urged, therefore, that the McNaghten rules should be broadened to make allowance for loss of control of one's actions owing to mental disease. In Western Australia (as well as in Queensland and Tasmania) this contention has been accepted. In this State the two McNaghten rules stated above are qualified as follows:

> A person is not criminally responsible if he is in such a state of mental disease or natural mental infirmity as to deprive him of the capacity to control his actions.

The modification of the McNaghten rules in this way means that little difficulty now arises in assessing the criminal responsibility of the definitely psychotic person. For all practical purposes, if a person is accepted as acting under the influence of a psychosis then he is no longer regarded as criminally responsible for his actions.

However, the difficulty confronting psychiatrists is coming from a different quarter. With the increasing knowledge of abnormal psychology among jurists, more cases of early mental abnormality are being referred for an assessment of their criminal responsibility. This is of course most desirable, but undoubtedly it is increasing the complexity of the psychiatrist's task. More frequently he is being asked to decide whether a delinquent has actually reached the stage of a psychosis or whether he must still be placed in that indefinite group known as the "psychopathic personalities". As you know, the term "psychopathic personality" has been used by different writers to describe varied types of mental abnormality. For the purposes of this paper the term is used to denote those individuals who show mental abnormality, particularly in their emotional reactions, but not to such a degree as to be actually psychotic, though of course in a number of cases psychosis may develop subsequently. The psychopaths then are the individuals who lie midway between normal and psychotic behaviour.

[1] Read at a meeting of the Section of Neurology and Psychiatry, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

The task of deciding whether a delinquent is actually psychotic can be a most difficult one. There are few things more chastening than to follow up the subsequent history of these individuals and to correlate developments with the initial diagnosis. I have therefore thought that it might be of value to present three case histories illustrating some of the problems which are met with.

CASE I.—A youth first came under psychiatric notice in 1942 at the age of seventeen years. He had been charged in the Children's Court with having unlawfully assumed control of two motor-cars and with stealing a revolver. It was recorded at his preliminary examination that he spoke in a rather "cheeky" and boastful fashion. He recounted his misdeeds without embarrassment and said that his main offences were concerned with the stealing of cars which he would drive at a high speed and usually leave in a damaged condition. Further investigation showed that he had what was recorded as a "shocking" history of delinquency dating from the age of five years, when he caused an extensive scrub fire whilst playing with matches. Apparently from this time on he was a constant worry to his parents, and at the age of eight years he was put in a reformatory school for stealing. From then on he was continuously being returned to the reformatory on some charge or other. At first stealing was the usual offence, but at the age of puberty he began to interfere sexually with young girls. At the age of fifteen years he held a job with a motor firm for about twelve months, learned to drive a car and became fascinated with speeding in motor-cars.

After a further period at the reformatory he joined the army when aged seventeen years, giving his age as nineteen years, but the brief period he spent in the army was filled with delinquency. He recounted with great glee how at a court martial he had "cheeked" Lieutenant-General Gordon Bennett. Eventually his true age was discovered and he was discharged.

On his discharge from the army he resumed his car-stealing escapades, which led to his appearance before the Children's Court. With a companion or two he would steal a car, pick up some girls and go for a joy ride, which would end either by his smashing up the vehicle or by his running out of petrol. The circumstances of these escapades show that he always endeavoured to escape detection and did in fact elude the police on a number of occasions.

He was kept under observation at the Reception House for approximately one month. It was recorded that there was no evidence of mental deficiency and that in fact he was rather alert and quick in many of his reactions. During the first two weeks he was fairly troublesome, interfering with other patients and indulging in "horseplay". After that he became more cooperative, but it was reported that he could not concentrate on useful employment for any length of time. Although he was seen by a number of experienced psychiatrists, none of them regarded him as psychotic and none was able to predict the ultimate outcome in his case. For want of a better label he was diagnosed as a psychopathic personality.

For the next five years his history continued much the same as before, periods of employment being interspersed with periods of imprisonment, although his offences were usually breaking and entering buildings.

Whilst in prison he conformed fairly well with prison discipline, except when he was "dared" by another prisoner to break some regulation, which he would then break out of bravado. In August, 1947, he was serving a sentence for burglary, when it was reported from the prison that he was becoming increasingly refractory. Psychiatric examination at this stage provided an explanation of his previous irresponsible behaviour. It was found that he was in a fairly typical state of mania with overactivity, flight of ideas and euphoria. He was transferred to the mental hospital and a course of narcosis produced a remission of his manic symptoms. An interesting feature was that he now claimed that he had been malingering throughout this attack of mania to secure his transfer to the mental hospital and so facilitate his escape. Investigation showed that he had indeed boasted to other prisoners that he was going to deceive the psychiatrists in this way. However, there seemed no doubt that he was suffering from a genuine attack of mania.

In the light of this development the previous reports from the Reception House of his "cheekiness", his "horseplay", his alert reactions and his inability to concentrate on a task for any length of time took on a new significance. A review of his case appears to leave no doubt that his delinquent behaviour from the age of seventeen years at least was based on his hypomanic personality. As his

delinquency prior to that age bore the same pattern, presumably it also had the same basis.

Although the problem presented in this instance was one of differentiating the psychopath from the hypomanic, a much more common problem in my experience is that of differentiating the psychopath from the schizophrenic. The following history illustrates the difficulties in this respect.

Case II.—A youth, aged eighteen years, was admitted to hospital in 1942, after being seen by a private psychiatrist, with a diagnosis of schizophrenia. It was reported that for the past month he had had auditory hallucinations, in that he claimed that he heard the noise of motor-cars and the voices of men outside his home, although nobody else could detect these sounds. His mother reported that he had given up his job a month before and would lie in bed most of the morning. She also stated that one day she had found him ironing some women's underclothes which had apparently been stolen. According to his mother he had been very difficult to handle since he was a child, although there was no previous history of actual delinquency. There was no family history of mental instability.

On admission to hospital he was very uncooperative and refused to discuss himself. After he had been examined by two psychiatrists at the institution, it was thought from the history and his general attitude that schizophrenia was the probable diagnosis. As he was very refractory it was decided to begin a course of "Cardiazol" therapy. After three convulsions he became very frightened of the treatment and absconded. It was not felt that the convulsant therapy had changed the mental picture in any way.

Five months later it was reported that he was in prison, where he had been sent when convicted of a number of offences mainly involving the stealing of women's underclothes. As he was proving very refractory, and as it was known that he had been in a mental institution previously, the authorities asked that he be examined by a psychiatrist. On this occasion he discussed himself much more freely. He claimed that he had been stealing the women's underclothing and selling it to a "fence", though whether this explanation was correct was not certain. However, he denied any fetishistic tendencies. He also denied ever having any hallucinations and said that he had simulated them to mystify his mother. His whole attitude at this time made the previous diagnosis of schizophrenia seem rather doubtful. For this reason it was decided not to certify him, but to give him the opportunity of coming to the mental hospital as a voluntary boarder for further observation. To this he readily agreed, and he was kept under observation at the mental hospital for six months. He was given no treatment, but was kept at useful employment. Throughout this period the opinion was strengthened that he was not a schizophrenic but a psychopath. Superficially he was well behaved, but he would take the opportunity of any relaxation of supervision to pilfer valuables from various parts of the institution. When detected in these misdemeanours he would fly into a fit of apparently uncontrollable rage. Although it was felt that he did have a poor control of his emotions, it was also believed that in part he consciously exaggerated these rages to evade discipline. At the end of his period as a voluntary boarder he was informed that he would be sent back to complete his sentence in prison, as he was considered responsible for his actions. He was told that a report to this effect would be made to the Crown Law authorities and that he must expect a prison sentence if he got into trouble in the future. It was impressed on him that he had not been certified as insane and that he had been given no treatment. For the remainder of his sentence his conduct in the prison was good. For more than two years after his discharge from prison he kept out of trouble, but towards the end of 1945 he was convicted of burglary. It may be mentioned that no tendency to fetishism was shown in this crime. He received a sentence of twelve months' imprisonment and during this sentence his conduct in prison was generally good. The main observation made was that he was rather easily led astray and needed firm supervision. In the two years since his last release from prison he has kept out of trouble as far as the police are aware. As it is now six years since he was first examined, everything seems to indicate that he is not a schizophrenic but a psychopath who appears to have responded surprisingly well to disciplinary measures.

Case III.—A man, aged thirty-one years, was arrested in 1945 on a number of charges of gross indecency with young boys. Whilst on bail prior to his trial it was reported that he was behaving in a peculiar fashion. He claimed that he had been vanquished by the devil, who was driving him to do evil things in an attempt to destroy his voice. He stated that his voice had exceptional qualities and that he would shortly become one of the greatest singers in the world. He said that he would not go to the pictures because he always felt that he should be the leading actor. He was seen by a psychiatrist, who considered that he was suffering from schizophrenia, and he was certified and admitted to a mental institution. After admission he claimed that he had been sent there to have his voice trained. He said that he felt that people were talking about him, though he had never actually heard them do so. It was found that he had been arrested for sexual misdemeanours whilst with the army in New Guinea, but the charges had not been proceeded with owing to his mental state and he was discharged from the army instead.

After he had been under the observation of two psychiatrists at the mental institution for about a week, it was felt that the diagnosis of schizophrenia was confirmed and it was proposed to report to the court accordingly. However, to explain his mentality at the actual time of committing the offences, it was decided to question him in a little more detail regarding the exact circumstances of the crimes. Rather surprisingly he said that he could not remember any of the details of the offences, that, in fact, he had a complete black-out for them. This was regarded as a puzzling feature, and, to stimulate his memory, some of the details alleged by a detective were read to him. Whereupon, instead of continuing with his story of a black-out, he proceeded to challenge many of the incidents mentioned by the detective. By now suspicions in respect to him were definitely aroused, and when he sensed this he became entangled in a tissue of contradictions. It soon became apparent that his symptoms of schizophrenia were simulated, and this was later confirmed from a reliable external source. At the subsequent trial, and also on his appeal against his sentence, the defence of insanity was abandoned completely. As there were certain bad features connected with his offences he received a lengthy sentence. During the ensuing three years no symptoms have appeared to suggest that he was ever suffering from schizophrenia.

Further investigation showed that his apparent mental disorder prior to his discharge from the army had also been simulated and that he had a long history of sexual delinquency dating back to the age of fifteen years.

Although more cases could be quoted to illustrate the different problems which arise, the histories presented will perhaps be sufficient to show how difficult it is for even experienced psychiatrists to arrive at an accurate diagnosis. Some of the difficulties met with having been outlined, it remains to suggest certain measures which may help to overcome them.

In the diagnosis of the cases under discussion the psychiatrist has to rely very largely on his clinical acumen. Special diagnostic procedures appear as yet to have only a limited practical application in the task of distinguishing the psychopathic personality from the early psychotic.

I have seen a limited number of patients given Rorschach tests, but in no instance were any significant clues furnished which had not been obtained from the usual clinical examination.

Whilst electroencephalography may have its uses in the diagnosis of epileptic states, it does not as yet provide reliable information for the diagnosis of early mania or schizophrenia. It is this last condition in particular which raises the most difficult problems.

The first task which the psychiatrist must accomplish is that of obtaining from the prisoner as accurate a psychiatric history as possible, particularly in relation to the offence alleged against him. In this respect a procedure which I have found of great practical value is to endeavour not to examine the prisoner prior to the preliminary hearing of the charge in the lower court. When this hearing has been completed one can then obtain the police depositions in the case, and in my opinion it is absolutely essential to obtain these and study them most carefully. It must be realized, of course, that these depositions are usually confined to the case against the prisoner. It has indeed been urged that studying them before hearing the prisoner's account may prejudice the psychiatrist. This disadvantage undoubtedly exists, but I think that, if it is recognized and guarded against, it is far outweighed by the value of approaching the very first interview with the prisoner with an accurate knowledge of the facts alleged against him. In my experience the first interview is the most crucial one. Quite often, prior to a second examination, an astute prisoner has had

time to draw deductions from the previous interview and is in a better position to confuse the psychiatrist. In any case, it seems to me that the psychiatrist must at some time study the lower court depositions before he arrives at the diagnosis which he will put before the higher court. My reason for saying this is that he must be able to correlate his diagnosis with any vital circumstances of the alleged offence. In one case that I recall, the prisoner claimed that he committed a capital charge whilst in a state of black-out due to epileptic automatism. On the other hand, a Crown witness in his depositions alleged that the prisoner had at the time of the crime attempted to prevent a third party from raising an alarm. In the particular circumstances of the case this apparently vital contradiction was capable of explanation, but it is obvious that a psychiatrist who was not aware of the discrepancy could find himself in a very embarrassing position.

Since the subject of black-outs has been mentioned, I should like to digress for a moment or two with reference to this question. I have no hesitation in saying that in a substantial majority of cases I have encountered in which a black-out has been advanced as the explanation of a criminal action, that explanation has not been a genuine one. It seems to me that the abuse of the black-out as a defence for crime is the greatest danger with which psychiatry from the medico-legal angle is confronted at the present time. This abuse not only represents a most serious obstacle to the proper administration of justice, but it constitutes a great danger to the confidence which should be placed in psychiatric evidence in the law courts. Nothing is more likely to weaken the confidence of the jurists in the psychiatrist than a too gullible attitude towards the black-out. I would urge, therefore, that, whenever this defence is put forward, particular caution should be exercised and every effort made to test its validity. I know of no better way of doing this than to lead the accused through the depositions, pointing out the circumstances which appear to invalidate his explanation. Quite often it will be found that the supposed black-out then assumes a curiously intermittent character; the accused will be able to remember incidents which count in his favour but not those which tell against him.

However, to pass on from this subject, one will find that, no matter how carefully prisoners are investigated, there will still remain a substantial number of cases in which it is extremely difficult for even the experienced psychiatrist to say with certainty whether a psychotic condition is the basis of the criminal behaviour existing. On the one hand you have the individual, such as in Case I, who remains in a state of incipient psychosis before the appearance of more florid symptoms elucidates the diagnosis. On the other hand you have the psychopath, such as in Case II, who does not develop a psychosis subsequently and may indeed develop a greater ability to conform with the law.

It would seem desirable that psychiatrists should have a keener realization of the difficulties of this problem so that they might press for a more satisfactory solution of it. Under existing arrangements the psychiatrist must recommend that these individuals be sent either to a mental hospital or to prison, although there may be serious objections to either course of action.

In general, the buildings and discipline of mental hospitals are not designed for the average psychopath. In consequence it is often impossible to keep him under proper control, and this has a disrupting effect on the staff and patients. More important still, the psychopath may become imbued and try to imbue others with the idea that he is no longer responsible for his actions and therefore has a licence to commit further crime. This is a very real danger and is not, I think, sufficiently appreciated by some who, out of sympathy for either the psychopath or his family, try to secure his admission to a mental hospital.

On the other hand, the idea of placing the incipient psychotic in prison on the same basis as the normal prisoner is distasteful. Under present conditions prison administrators must enforce the same discipline for all inmates. Furthermore, owing to the absence of continuous psychiatric supervision, a psychosis may develop considerably before it becomes obvious to the prison staff.

The most practical solution of the problem would seem to be the establishment of a special section of the prison department where all prisoners who appeared to need psychiatric investigation or supervision could be placed. The best method of organizing such a section of the prison system has been described by Norwood East in the Home Office pamphlet "The Psychological Treatment of Crime". It will suffice to emphasize that the psychiatric supervision provided would have to be adequate, not only quantitatively but above all qualitatively. Enthusiasm is a dangerous substitute for experience in this particular branch of psychiatry.

The establishment of a psychiatric division of the prison should also contribute materially towards the more satisfactory assessment of criminal responsibility prior to trial. This would no longer be determined by a visiting psychiatrist forced to rely upon reports from staff without psychiatric training. The prison psychiatrist would have adequate opportunity to observe the prisoner and would have the assistance of staff with the necessary training. The more I see of these borderline offenders, the more I am convinced of the need for such a psychiatric section as part of every prison department.

THE TREATMENT OF NEUROSYPHILIS.[1]

By L. B. Cox,
Melbourne.

Introduction.

Syphilis of the central nervous system, although in our days by no means the commonest nervous affection, remains an extremely important disease. I will set out for you some of my experiences in its treatment which have extended over a good many years, although when I come to the important recent advance, penicillin therapy, I must speak largely by the book. There will be little new in what I will say to you, and I will be unable to deal with the treatment of individual symptoms.

I need hardly remind you that changes in the cerebrospinal fluid have been found as early as the primary stage of syphilis and that therefore the infecting organism can invade the central nervous system at an early stage of the disease. Also you will recall that the spirochæte can seemingly remain latent there or elsewhere in some form, in a way not clearly understood, to produce the serious manifestations of neurosyphilis as long as forty years after infection.

The convenient clinical and pathological division of the disease into meningo-vascular and parenchymatous types is by no means absolute, and both forms usually exist together, although one or other type is likely to predominate. As Kinnier Wilson reminded us, "syphilis of the nervous system is one and indivisible". Yet at an early stage it is usually an infection of the vascular tree, and through it of the meninges, producing an arteritis and a perivascular gummatous process, with or without meningitis. At a later date it can penetrate the vascular barrier, to invade the neurones directly. So in general paralysis of the insane will be found a diffuse syphilitic encephalitis with involvement of vascular tree, meninges, neurones and neuroglia. The position in tabes is not so clear. Whether the fundamental lesion is an inflammation of the radicular nerve of Nageotte—that part of the dorsal root lying in the subarachnoid space, but lacking a pial covering—has not been certainly proven; and the infecting organism has only rarely been found in association with visible lesions. The causation of the other so-called parasyphilitic disease, optic atrophy, is also not clear. Of late there has been a suggestion that it is less a primary toxic degeneration than a direct invasion of the

[1] Read at a meeting of the Section of Neurology and Psychiatry, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

nerves and tracts. If so, some cases can be singularly resistant to the form of treatment usually effective in meningo-vascular syphilis.

In the short time allotted I will pay most attention to the late manifestations of neurosyphilis. Firstly, I will describe the plan of campaign used in my clinic before the penicillin era, in which period most of the cases analysed fall. Before so doing I must make it clear that the plan has never been strictly adhered to. Each patient presents a unique problem, in which clinical, psychological and social factors all call for the modification of any set plan by common sense and circumstances.

Briefly, the agents available for treatment have been the metals mercury, arsenic and bismuth, and potassium iodide. Later the antibiotic penicillin was added.

The plan has been as follows: (i) iodide and perchloride of mercury given orally for from one to two weeks; (ii) pentavalent arsenic added to this, usually as tryparsamide given intravenously in eight weekly injections (one of one gramme, one of two grammes and six of three grammes); (iii) twelve weekly intramuscular injections of a bismuth preparation, usually "Bismol", mercury as a rule being omitted. At the end of the course the blood Wassermann test is again carried out and the course repeated. At the end of the second course the cerebro-spinal fluid is retested. Such courses have been repeated, according to circumstances, sometimes for several years.

The following modifications have been at times necessary: (*a*) substitution of pentavalent "Acetylarsan" for tryparsamide in children and in some adults with small veins; (*b*) the use of trivalent arsenic when visual changes have occurred with the pentavalent form; (*c*) transference of the patient to a mental hospital for malaria treatment when the mental state of the patient has been incompatible with treatment at or in a general hospial; (*d*) transference for malaria treatment when a patient with mental disturbance has not responded to one course of tryparsamide; (*e*) the use of penicillin early for all cases of neurosyphilis, since it has been available, as also for such older cases as have not responded satisfactorily clinically or who have been "Wassermann-fast". It has particularly been used in cases of optic atrophy or when pentavalent arsenic has affected vision.

Analysis of Cases.

The series is a consecutive one of 60 cases. Reference, too, will be made occasionally to some special case which has not fallen into this group. Time has not allowed the collection of a larger series.

General Paralysis of the Insane.

Of this series, 17 patients were suffering from general paralysis of the insane; their period of observation has extended from a minimum of four to about twenty years. No recent case has been included. Others have not been included, as the patients have been immediately transferred to mental hospitals and not heard of further. Four patients who have returned after malaria therapy have remained well, except two with tabetic symptoms which have not been relieved.

The effect of malaria treatment on this disease is well known from several large groups of cases. Briefly, less than 50% of patients are able to return to a useful occupation, although in rather more than 50% the disease is arrested or remits for a useful period. The immediate mortality is just under 10%, while 40% of the treated patients die within five years. I have nothing to add to this aspect of treatment, as those returned to me have, of course, been successfully treated.

Initial treatment with tryparsamide was forced on my hospital early. Two patients given malaria proved unsuitable for detention in the general wards available, and difficulties were raised to further admissions. I therefore will analyse the 13 cases in which tryparsamide was given initially. Of these I will omit one of two in which I gave malaria, which happens to fall into the group under analysis. Malaria was employed, not because the patient did not respond to tryparsamide, but because, at the time, we intended to use it as a routine form of treatment. He has had malaria on several occasions, as his infection is of the relapsing type. Although the results of serological tests of blood and cerebro-spinal fluid had become negative, it was recently again necessary to commit him to an institution. It is now over twelve years since his original course.

Of the remaining 12 patients given tryparsamide first, five have not required malaria and all are well and in employment. One, still under observation, had a minor relapse after the first course, but not since. He has been well now for about four years. The others have remained well for periods from four to fifteen years. Three others responded well to tryparsamide, but ultimately relapsed and were given malaria. Two of these had been controlled for three years by tryparsamide, although one suffered several relapses immediately influenced by tryparsamide therapy. Finally, each patient's condition became resistant to tryparsamide and he was given malaria. In each case the condition proved resistant to this also, one patient dying within four months and the other within a year. The third case was interesting, in that the patient remained well for nine years, when the results of his blood and cerebro-spinal fluid Wassermann tests were negative and his gold curve, once of the paretic type, had become normal. A few months later he apparently relapsed and was given malaria. Afterwards he was well, his blood and cerebro-spinal fluid both being still normal.

Four patients did not improve with tryparsamide, three after one course and one after two. Of these, three were transferred for malaria therapy, but the condition proved resistant, two dying in hospital. One improved sufficiently to be discharged from hospital, but was readmitted after seven months; his condition proved resistant and he is now undergoing progressive dementia in hospital. The fourth, whose condition was mild, with well-marked pyramidal tract signs, responded to malaria, and he is now well after several years, except for his paraplegia.

It is suggested from this small series that as a rule neurosyphilis proving resistant to tryparsamide proves resistant to malaria. It must also be pointed out that, whereas tryparsamide was often given without malaria, malaria when given first was usually followed by tryparsamide.

Although such a small series can hardly be subjected to statistical analysis, figures derived from other clinics of patients treated with malaria and tryparsamide would hardly lead one to expect a better response than this group treated by tryparsamide alone. Nevertheless, it seems likely that the combination does give better results on the whole than the treatments used singly.

As regards penicillin, my own use of this in general paralysis of the insane is too recent and small to be of interest. However, the large American series and one small English one published by Purdon Martin show its unquestionable value. It is certain that it is at least as effective as malaria or tryparsamide. It will, however, be observed that in certain cases the disease resists all forms of treatment, but whether some infections are more resistant to one form of treatment than another is not certain. My own practice at the moment is to combine penicillin with the older treatment. I would use initially penicillin, followed or combined with tryparsamide, and would advise malaria if the clinical response is unsatisfactory.

Tabes Dorsalis.

Despite statements that antisyphilitic treatment is ineffective in arresting *tabes dorsalis*, I have no doubt at all that it is of value in most cases and of great value in some. To withhold it where there is an increase of cells in the cerebro-spinal fluid would in my judgement amount to negligence; where the fluid is normal there is more justification. I would entirely agree with Kinnier Wilson when he stated that "the sooner tabetic cases undergo vigorous treatment, the better".

I will confine my analysis to 23 cases, a consecutive series, excluding cases of optic atrophy. In these almost all patients were given tryparsamide and five were given malaria.

Two of these patients died. One responded well symptomatically to a course of tryparsamide, but then developed

a severe infection of the urinary tract to which he succumbed. The other did not respond and developed serious mental changes. She was sent for malarial treatment, but died.

Before accepting a defeatist view of this condition we can profitably examine the remaining 21 cases. The majority of the patients examined had an increase of cells in the cerebro-spinal fluid and an increase of protein. Almost invariably such fluid soon returned to normal as regards these constituents. This of itself is surely justification for treatment, even without symptomatic benefit, and I cannot believe that the return in these cases was fortuitous. At least the disease pathologically viewed showed considerable diminution of activity.

Now as regards symptoms, of which the chief are pain and ataxia, these may be considerably lessened by treatment if the patients are to be believed. Of the 21, 15 claimed relief varying from a lessening of pain to complete cure. Patients not improved included some with Charcot's joint, which, in this series and in several other cases not included, responded little if at all to antisyphilitic treatment. In other cases lightning pains persisted, but I can recollect only one case, not included in this series, in which tractotomy was necessary for the relief of pain.

I must admit to little experience in the effect of malaria alone, but so far I have not been impressed by its advantages over tryparsamide. Five patients of this series were given malaria, two at the onset because they were suffering from taboparesis, and three for symptomatic reasons. In the first two cases paretic symptoms were most satisfactorily relieved, but not tabetic symptoms; neither patient responded later to tryparsamide therapy, their pains persisting. Three others were given malaria on account of persisting pains, but were not relieved. Nevertheless, it is quite certain that malaria will usually produce excellent results from the pathological standpoint, as judged by examination of the cerebro-spinal fluid. I do not know, however, that it is more effective than tryparsamide. It is certainly of great value in replacing tryparsamide in cases of optic atrophy, as this drug itself can seriously affect vision.

I have had some experience of penicillin in tabes, but all of my patients, except two not considered here, had had previous tryparsamide therapy. On two occasions the result of serological examination of the cerebro-spinal fluid, which was weakly positive, has become negative, in one case to become positive again and not to be altered by the further administration of penicillin. So far it has not helped the symptoms in the resistant case to any conspicuous degree. I would certainly use it first of all in any new case and have done so twice, but with tryparsamide therapy. The response so far has not differed from what one might expect with tryparsamide therapy alone.

Optic Atrophy.

Optic atrophy is said to occur in about 20% of tabetics, although it has been rather less frequent in my experience. The series, however, includes three severe cases. One of the patients affected had previously had malaria without symptomatic benefit, although his cerebro-spinal fluid had become normal. Treatment was continued with "Maphasen" and bismuth, but he deteriorated. Another patient improved with the same treatment sufficiently to play in a "champion of champions" bowling contest, but later suddenly deteriorated and rapidly became blind. The third patient had congenital syphilis and did not improve with therapy.

It is fairly certain that penicillin with or without malaria is the treatment of choice. I have had one patient with severe optic atrophy since penicillin became available who did not improve with bismuth and "Mapharsen", but who after receiving penicillin regained visual acuity of $^{6}/_{12}$ in the less affected eye; this had previously been $^{6}/_{60}$. The result of a blood Wassermann test too became negative.

Meningo-Vascular Syphilis.

The treatment of meningo-vascular syphilis is considered more satisfactory than that of the later manifestations of neurosyphilis. Thus it may respond well to mercury, bismuth, the iodides and trivalent arsenic. This improvement can usually be fairly confidently expected, provided it is not expected that such lesions as a severely damaged spinal cord will recover or that a dense mass of pachymeningitis will resolve. Seventeen cases occurred in this series. Some of the patients were treated with trivalent arsenic, but the majority with the pentavalent form. Although the latter is regarded as having less systemic effect than the other form, it frequently seemed to me to act as favourably, judging by the number of patients in whom the result of a blood Wassermann test became negative. Favourable results were observed in many types, such as acute meningitis and vascular thrombosis. Unfavourable results were particularly observed in chronic pachymeningitis of the cord and brain. In two cases of spinal pachymeningitis, only one of which is included in this series, antisyphilitic treatment had little effect. Each patient was operated on and the cord freed, but the results were most unfavourable. In another case of pachymeningitis of the summit of the brain the patient died in *status epilepticus* during treatment, although he appeared to be progressing favourably. In our one case of Erb's paraplegia the patient was unaffected by antisyphilitic treatment.

Congenital Syphilis.

Congenital syphilis should be treated as is the acquired form, and I have seen favourable results ensue. However, in this series only three cases occurred. One of the patients was asymptomatic, but his cerebro-spinal fluid yielded a positive Wassermann reaction. In one case general paresis was seemingly arrested and in one tabes was not relieved. I have had no experience of penicillin in such cases, which are rare in this country, but I would immediately use it in any new case.

Summary.

1. An analysis is made of sixty cases of neurosyphilis treated over many years. The effectiveness of tryparsamide or malaria is noted for about 50% of cases of general paralysis of the insane. Penicillin is said to have at least comparable effects.

2. It is concluded that the majority of patients with *tabes dorsalis* are benefited symptomatically by tryparsamide therapy combined with bismuth. Even in those cases in which this benefit is not obtained the disease may be arrested from the pathological standpoint.

3. The majority of patients with meningo-vascular syphilis respond well to either trivalent or pentavalent arsenic. Patients with chronic pachymeningitis, with Charcot's joint or with Erb's paraplegia are resistant to treatment with metallic drugs.

RECENT ADVANCES IN APPARATUS USED FOR ELECTROCONVULSANT THERAPY AND ELECTRONARCOSIS.[1]

By LINDSAY WILSON, B.A. (Melbourne), M.B., Ch.B. (Edinburgh),
Perth.

Electroconvulsant Therapy.

MOST people are aware that electroconvulsant therapy was first introduced by Bini and Cerletti in 1937, using a set named "The Solus". This was followed by the Edison-Swan machine, designed by Grey Walter, and the MacPhail-Straus apparatus as used at Saint Bartholomew's Hospital from early 1939. Scores of others appeared all over the world, but all were fundamentally the same, designed in various ways first to measure transtemporal resistance and then to deliver alternating current at a selected voltage for an accurate period of time in tenths of a second.

[1] Read at a meeting of the Section of Neurology and Psychiatry, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

Until recently little or no attention was paid to wave form. It was known to be sinusoidal, but no one ventured beyond theory into the finer points of electronics.

Gradually, in other fields of medical electrotherapy it was noticed that steeper wave forms produced less disturbance to cortical neurones and less physiological excitation. Condenser discharge is the ideal way to produce a steep-fronted wave, but the necessity of working off direct current in many cities led to the utilization of the argotron valve. This gave a much steeper leading front than normal alternating current, but the energy factor still remained uniform for each impulse.

The rapid drop in inter-electrode resistance during the passage of the current suggested that a rapidly damped train of waves would be preferable to the massive uniform bombardment of the brain which all these sets were giving. It was beginning to be seen that even one-tenth of a second was a very long time electrically speaking.

If a higher voltage could be given for the first cycle or two, the current could advantageously taper off very rapidly. Moreover, the higher initial current would ensure the convulsions and the brain would sustain minimal damage. It was known that the brain could withstand 2000 volts if applied only for a split second.

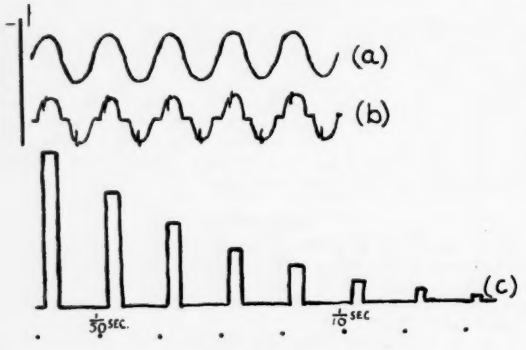

Figure I.

(*a*) Sinusoidal wave, (*b*) modification with "Argotron" valves, (*c*) condenser discharge (monophasic).

At "Barts" work was resumed on the condenser discharge principle, which, in addition, had the advantage of monophasic or diphasic discharge; the former was a very safe current suitable for children and hyperpietics, whilst the latter was strong enough to ensure convulsions in even the most resistant.

Finally, the technical difficulties were overcome and, moreover, a universal circuit suitable for use from alternating current, direct current or a 12-volt battery was designed, the weight being only 25 pounds.

Oscillograph tracings showed that this unit was theoretically ideal.

Clinically it was an advance for the following reasons. (i) No preparation of the patient was necessary. (ii) The surface area of the electrode could be reduced to a one-inch diameter, which made possible more accurate localization of the current's path through the frontal lobes and less radiation to medullary centres. (iii) Dosage could be calculated in absolute units of energy (joules), for this wave knows nothing about transtemporal resistance. As a rule a dose of 15 joules is given. This is approximately equal to 300 volts for one-fiftieth of a second. (iv) Every patient would almost certainly undergo convulsions in the first instance, even if saturated with barbiturates.

Clinical trials confirmed all these points, and it was observed that the convulsions were less violent and over sooner; recovery was quicker and memory disturbance less; muscle strains (due largely to pre-shock anxiety and tension) were diminished; and more patients could be treated per hour. We never allowed a patient to witness any stage of treatment or recovery of another patient at "Barts".

This apparatus is known as "The MacPhail-Strauss Electroplexy Unit" or simply "The Plexacon". It is now widely used throughout Europe and costs £80 sterling in London. It is fully described in *The Lancet* of December 21, 1946. Electroconvulsant therapy is still cardinal for depression and is rarely used for any other condition.

Electronarcosis (or Electrocoma).

Towards the end of the war work initiated as early as 1902 by Leduc was revived with a view to producing a state comparable with insulin coma by means of electricity. A constant but controllable alternating current of up to 300 milliamperes was required, and this was easy enough to design.

In electronarcosis (or, probably more correctly, electrocoma) no convulsion occurs. The application of the current (usually 160 milliamperes) renders the patient immediately unconscious. Strong muscle spasm with opisthotonus occurs resembling decerebrate rigidity. Respiration is arrested, the heart beat is inhibited, and the blood pressure becomes alarmingly elevated. After thirty second the current is dropped to about 65 milliamperes, whilst respiration is reestablished. A mixture of carbon dioxide and oxygen is administered. The current is then quickly increased by five milliamperes every fifteen seconds until a mild respiratory stridor develops, whilst the limbs remain in a state of tetanoid contraction. This is kept up, if possible, for seven minutes.

Surprisingly enough, the patient recovers consciousness and reorientation more quickly than after electroconvulsant therapy, but feels more exhausted. Premedication with atropine (one seventy-fifth of a grain) is usual.

The Shotter-Rich apparatus was one of the first to be used in London and was shortly followed by one designed by MacPhail and Strauss. Both gave excellent results, but the treatment was undoubtedly far more dangerous than electroconvulsant therapy and needed a strong team of doctors.

At "Barts" we were not happy about this raw alternating current, its violent physiological upset and its danger of cardiac and/or respiratory collapse. Moreover, if the patient "woke up" after the initial thirty seconds he would rarely consent to continue the treatment. Some physicians gave an intravenous injection of barbiturate, but we felt it advisable not to mask any of the effects. Consequently during 1947 we proceeded very cautiously indeed. Nevertheless the results with the patients who continued the treatment were so encouraging that attempts were made to design a unit which would give a far more refined current. Inspiration came from radar, and a similar pulsating current variable in respect to voltage, pulse repetition frequency and pulse width was produced in December, 1947. Its success was immediate. The undesirable physiological reactions were minimal and it was apparent that now we had apparatus which would deliver a current which was both safe and effective.

Treatment is commenced with one kilovolt at a pulse repetition frequency of 50 pulses a second and a pulse width of 10 microseconds. After thirty seconds the voltage is reduced to 0·4 kilovolt. Breathing rhythm resumes spontaneously and no oxygen is necessary. Pulse width is dropped to five microseconds and then voltage is increased to 1·1 kilovolts and by slower steps to 1·25 kilovolts. Meanwhile the pulse repetition frequency is increased to 80 pulses per second and the pulse width further decreased

Incidentally the actual current is usually six milliamperes, as opposed to 250 milliamperes with the other machines.

This procedure of increasing the kilovolts and the pulse-repetition frequency offsets any tendency to conditioning of the cortex, and this prevents the coma from becoming "light".

The shortening of the pulse width gradually eases the stress on the heart and increases relaxation, which, in time, because of the slower accumulation of waste products of muscle action, reduces oxygen deficiency. These various factors enable one to control cyanosis and respiratory and

cardiac strain and, at the same time, to minimize the tetanoid state.

Treatments are continued until clinical improvement is maintained.

The apparatus is to be known as "The MacPhail-Strauss Electrocoma Unit" or simply "The Pulsacon".

It will not go into production until a sufficient number of patients have been treated and adequately followed up.

So far, three types of subjects are being selected, provided that the history is fully known in every case: (*a*) those with severe manic-depressive psychoses not responding to electroconvulsant therapy, (*b*) degenerating schizophrenics, and (*c*) those with severe obsessional-compulsive "psychoses"; in other words, those with conditions in which prefrontal leucotomy is normally indicated. The question of electronarcosis replacing routine insulin treatment in acute schizophrenia is, as yet, undecided.

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GOLD THERAPY IN RHEUMATOID ARTHRITIS.[1]

By L. J. A. PARR,
Sydney.

DEFINITION OF NOMENCLATURE.

IT is necessary to define what is meant by the term "rheumatoid arthritis" because of the inclusion by some of ankylosing spondylitis under this heading.

The New York Rheumatism Association includes ankylosing spondylitis under this term, and the American Rheumatism Association does the same, as is evidenced by the following in their classification: "Arthritis, Rheumatoid. Specify as Multiple or of Spine." Matthew Ray, an English rheumatologist, defines rheumatoid arthritis as "a systemic disease, mostly, but not entirely, affecting women during the third and fourth decade, and is characterized by progressive and symmetrical change in the joints and peri-articular tissues, usually beginning in the smaller and extending to the larger joints. It is accompanied by varying degrees of pyrexia and constitutional disturbance, and is often preceded by vaso-motor and sensory phenomena in the affected limbs".

Most authorities, especially the American, include focal, infective or metastatic arthritis in the rheumatoid group. We will include these in this article as well as Still's disease (juvenile rheumatoid arthritis).

PROTEAN CLINICAL PICTURE OF THE DISEASE.

Since the rheumatoid syndrome is associated with much variability of signs and symptoms, the diversified picture of the disease must be kept in mind. No one who has closely followed the disease for many years and carefully watched and followed his patients can fail to appreciate the diversity of the rheumatoid pattern. The one basal pathological structure producing disturbance of the locomotor system does remain; but the superimposition of diversified characteristics makes hardly any two consecutive cases alike. In some, muscular spasm and hypertonicity are well marked, whilst in others pronounced hypotonia is present; in some cases the hands are cold and clammy, with distended venules and pink finger-tips, the ears, nose and lips being frequently pale and slightly cyanosed, whilst in others the hands are dry and there is no facial or ear pallor. The vasoconstrictive phenomena are common, but by no means universal; they may occur in the presence of a greatly raised or almost normal erythrocyte sedimentation rate, but are generally very prominent if hyperthyreoidism is an accompaniment. In some cases the periarticular tissues are mainly involved, whilst in others the main weight of the disease falls on synovial membrane, articular cartilage and subchondral bone. Some joints will ankylose even if not immobilized by splinting, whilst others will not ankylose if immobilized for long periods. In some patients muscular atrophy and loss of weight are pronounced, whilst in others they are almost negligible.

In rheumatoid arthritis associated with the menopause hypothyreoidism may be present in varying degree, whilst in the younger women the symptoms and signs may resemble hyperthyreoidism without or rarely with a raised basal metabolic rate.

If we keep in mind the variability of the pattern of the disease, the limitations of any one remedy will be more easily understandable and the need for a therapeutic advance on a wide front readily conceded.

VARIABILITY OF THE BIOCHEMICAL PATTERN.

Rheumatoid arthritis may be associated with a great disturbance of the plasma proteins, a reversal of the albumin-globulin ratio, a much-increased amount of euglobulin and fibrinogen, and a total amount of protein which is normal in the early stages of the disease and lowered as the disease increases in severity and chronicity. Liver dysfunction has been revealed by a number of tests, chiefly the serum colloidal gold flocculation test, the result of which is positive in over 50% of cases.

GOLD THERAPY.

We can, therefore, approach the subject of gold therapy knowing the clinical condition with its variability and the marked systemic disturbance associated with many cases.

The use of gold therapy in rheumatoid arthritis was proposed by Feldt, and Forestier, one of the first and most ardent advocates, reasoned that if gold was active in a chronic disease like tuberculosis it might be of value in another chronic infective disease such as rheumatoid arthritis. Gold therapy suffered much from the super-optimism of its early advocates, but also from the pessimism of some of its early users. It is still used extensively after twenty years of trial and error, and most rheumatologists, "with few exceptions, report results therefrom better than those obtained with any other single remedy" (Hench *et alii*).

Active Gold Salts.

Active gold salts contain a high proportion of gold (40% to 60%) combined with a sulphur radical. Colloidal gold preparations, including the sulphide, have been abandoned by most clinicians. Coke, of the Charterhouse Rheumatism Clinic, used six preparations over a number of years and places them in order of therapeutic efficiency, from the weakest to the strongest, as follows: colloidal gold sulphide, "Auric Succinate", "Novacrysin", "Myocrisin", "Solganol B Oleosum" and "Auro-Calcium".

The gold salts most commonly employed in Australia are:

Chemical Name.	Proprietary Name.	Gold Content.
Gold sodium thiomalate	"Myocrisin"	45% to 47%
Gold calcium thiomalate	"Auro-Calcium"	46%
Gold thioglucose	"Solganol B Oleosum"	50%

"Sanocrysin" (gold sodium thiosulphate) is not commonly employed and I have abandoned the use of "Lopion"

[1] Read at a meeting of the Section of Medicine, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

(sodium auro-thiosinamine benzoate). "Auric Succinate" (Crookes) has been used by Coke, but he states that the general impression at their clinic was that this compound, which does not contain sulphur, is not as efficient as those containing sulphur and gold together. This compound has been employed latterly because gold sodium succinimide (containing no sulphur) has been found effective in curing experimental arthritis in mice. Because of the ease of aqueous administration I have abandoned the use of preparations in oil and find no increase in complications.

Hartfall and Garland found "Lopion", a gold preparation for intravenous administration, of very low toxicity, but this view is not shared by all.

Indications for Gold Therapy.

Comroe emphasizes that there are three main clinical difficulties confronting the physician treating rheumatic subjects with gold salts, the first important one being the difficulty of knowing how to choose the right patient and of avoiding those who will not respond, as gold does not benefit all subjects of rheumatoid arthritis. Coke states that the differential sedimentation rate test helps to define the subject suitable for gold therapy, but most rheumatologists judge only by experience and trial and error. There is no doubt that some physicians use gold therapy only when all other methods have failed, but many now use gold in the early stage before irreparable damage has occurred. Many American rheumatologists have swung over to this point of view. Some American authorities go so far as to say that it should be used early and in every case, but a few are still hostile to its use and claim just as good results by the older methods employed. Let us remember that, if the older methods used previous to the advent of gold therapy were efficient or just as efficient, gold therapy, because of its toxic effects, would have almost certainly died a natural death, especially after twenty years of clinical trial. There is no doubt that patients with the low-grade periarticular type and the mixed type of arthritis are generally rapidly helped by gold and that those with marked vasoconstrictive evidence, such as pallor and cyanosis, are frequently unaffected by gold. It is in these cases that albuminuria and blood dyscrasias are liable to develop, and it was in such a case that Dr. Shipton and I reported a fatality, although at that time the dosage employed was much greater than that in use today. The most important indication for gold therapy is the failure of the disease to respond to other therapy. I have found it of great value when the sedimentation rate is normal, especially in subjects with an associated syndrome like Raynaud's, but also in those patients with a greatly raised sedimentation rate, in the vicinity of 100 millimetres per hour (Westergren). In those cases in which a dry inactive skin is seen, as opposed to a moist clammy pale skin with cyanosis, gold therapy is a very valuable remedy, and the presence of psoriasis is no bar against its employment.

Age is no barrier, as a child with Still's disease may improve just as well as an old person of seventy years.

It has been said that the later the employment, the less the chance of effecting improvement, but in many cases improvement can be considerable even though the disease is of years' duration. Let us observe that it will not regenerate eroded or destroyed articular cartilage.

Contraindications.

Contraindications are severe *diabetes mellitus*, nephritis, ulcerative colitis, hepatic insufficiency, blood dyscrasias and hæmorrhagic tendencies, whilst some would ban any allergic subjects. Pregnancy and a history of exfoliative dermatitis are contraindications, as well as rheumatic fever with or without carditis and disseminated *lupus erythematosus*. I have used gold in cases of asthma without any adverse reactions. We do not employ gold for patients with associated severe hypoprotinæmia or with a reversal of the albumin-globulin ratio. Severe secondary anæmia and a low leucocyte count also contraindicate gold therapy.

Dosage and Method of Administration.

Whatever preparation is employed, it is essential to begin with small dosages (10 milligrammes) and increase slowly to a maximum dosage of 50 milligrammes of the gold salt. This method ensures a minimum of reaction in that case, which none can foretell, in which after one or more doses the patient will suffer from exfoliative dermatitis. After an experience of gold over a period of twelve years I can affirm that safety lies only with small dosage. Further, since employing these smaller doses, which Dr. Shipton and I began in 1937, we have never had a fatality and rarely any severe complications. In some cases it has been noted that one preparation will provide improvement where another has failed, but I have found very little difference between any of the three main preparations used in Australia. Children are given a dosage commensurate with their age.

Our procedure over the years has changed from the older method of giving a course of twelve injections followed by an interval of six to eight weeks, then another course and so on, to continuing therapy weekly till improvement occurs, as measured by the sedimentation rate and the clinical condition.

The injections are then spaced out, two weeks being left between and later three weeks, till finally four or six weeks' intervals are maintained, the determination of the space between injections being dependent on the clinical condition and the erythrocyte sedimentation rate. This procedure has been followed in children as well as in adults.

This maintenance dosage has been employed by us during the past three years and a number of patients are under such control. It is significant that many rheumatologists in both England and America have adopted this method. Ragan and Tyson state: "We now treat patients with rheumatoid arthritis with a maintenance dose of gold, continuing 50 mg. every two or three weeks indefinitely." However, many clinicians still adhere to the routine used initially, namely, a course of twelve doses followed by an interval of six to eight weeks. Forestier laid down the rule that "whatever gold salt is employed, treatment should be carried out by a regular series of doses, with intervals for rest during a period of at least one to two years".

He also stressed early treatment and regular blood examinations. We check the erythrocyte sedimentation rate and make a full blood count every three months, and later, after improvement, every six months or even up to one year.

In one of our cases eight courses have been given over a period of five years with varying intervals between the courses, the time being determined by relapse after therapy was completed, but in another, in which maintenance therapy has continued over four years, no relapse and no complications have occurred. In this latter case the interval between injections is six weeks, each dose being 50 milligrammes of aqueous "Myocrisin".

Forestier has seen cases in which a patient who did not respond to small doses improved with large, and some in which one type of salt was inactive and another successful. However, Ragan and Tyson in a series of cases (142) followed over a number of years (a maximum of six) found no significant difference in the toxicity and response to therapy whilst employing "Myocrisin", calcium aurothiomalate and "Solganol B Oleosum".

Some English rheumatologists are still using large doses up to 0·2 gramme, after commencing with small doses, but in our opinion and experience over twelve years larger doses will not produce amelioration if doses of 0·05 gramme or 0·1 gramme (rarely used) are not productive of relief, and the risk of toxic complications is greatly enhanced by the larger dosage.

Freyberg "found that weekly injections of 50 mg. of gold salts were just as effective as doses of 100 mg. but that weekly injections of 25 mg. gave inferior results". There would seem to be a minimum requirement for therapeutic efficiency and most clinicians agree as to 50 milligrammes being the suitable effective single dose. This dosage can, therefore, be considered to be an efficient

therapeutic dose with minimal toxic effects. As the trend has been away from larger single doses, it has been towards small dosage over very long periods (years) and we see fewer references in literature to the total dosage of one gramme per course as arbitrarily laid down in the earlier literature on gold therapy. Some of our patients are given monthly injections of 0·05 gramme of "Myocrisin", that is, 0·65 gramme in one year, and others a similar dosage every six weeks, a total of 0·45 gramme per year. Hench *et alii* state that the plan of continuing maintenance doses has now been widely adopted.

Small dosage and maintenance therapy are definite advances in modern chrysotherapy, on the one hand minimizing toxic reactions without any loss of therapeutic effectiveness and on the other diminishing the incidence of relapses. The fact that rheumatoid arthritis does burn itself out in many cases by the efflux of time renders imperative the need for long-continued effective control if possible of attainment. Gold therapy should be maintained until the erythrocyte sedimentation rate is normal and all local areas show no signs of activity. Morning stiffness is evidence of activity.

Mode of Action.

"Myocrisin" is bactericidal *in vitro* against certain streptococci and renders the serum bacteriostatic against these organisms.

On the pleuropneumonia-like arthritis of mice gold compounds exert a therapeutic effect. It is the combination of gold and organic sulphur which produces therapeutic efficacy, the opinion being held that the SH radicals combine and exert their therapeutic action as gold glutathione or gold cystein complex compounds.

Gold accumulates in the body, 80% of the dose remaining after one week. It circulates in the blood, intimately bound with the serum protein, and concentrates in the synovial membrane but not the synovial fluid.

Toxic Reactions.

Toxic reactions may occur after a few injections of small dosages (such as 0·025 gramme), at the end of a first course, after a number of courses have been given or months after gold therapy has been completed. In one of our cases it occurred with small dosage (0·025 gramme) after the second dose injected.

Skin.

The skin is the commonest site of reaction (60% or 70%), and various types have been observed, from a mild to an exfoliative dermatitis. Macular, scarlatiniform, erythematous and urticarial rashes have been noted.

Mucous Membranes.

Loss of taste, anæsthesia of the tongue, stomatitis, dysphagia, superficial vulvitis often accompanied by furunculosis, conjunctivitis and keratitis have been observed.

Alimentary System.

Diarrhœa, vomiting, hiccup and jaundice may occur, the latter being one of the most serious complications which can occur and causative of a number of deaths.

Renal Complications.

Albuminuria is fairly common, but permanent kidney damage rare. In one of our cases this did not clear up for six months. It precludes further therapy.

Pyrexia.

So-called gold influenza may occur with a temperature of 101° F. or higher. It precludes any further use of gold. In one of our cases it occurred with a dose of 0·025 gramme of "Myocrisin".

Respiratory System.

A chronic cough may occur without any physical or radiographic changes—the so-called gold bronchitis. It is of very little significance.

Central Nervous System.

Gold has been found in the cerebro-spinal fluid after parenteral administration. Deafness has been reported.

Hæmatopoietic System.

Amongst the most toxic effects of gold have been those on the bone marrow and circulating blood. Aplastic anæmia, thrombocytopenic purpura, agranulocytosis and *aleuchia hæmorrhagica* have been reported as due to gold salts.

Forestier reported two cases of agranulocytosis and two of purpura in 500 cases.

Hartfall and Garland reported one death from aplastic anæmia in a series of 274 cases; Parr and Shipton reported a fatal case of *aleuchia hæmorrhagica.*

The mortality rate in all reported cases has been about one in every 250 cases, or about 0·4%.

Incidence of Reactions.

The percentage of reactions has greatly declined since small dosage has been employed, and Dr. Shipton and I have had only one death, which occurred amongst the first 70 cases reported in 1937, but none since, and well over 500 patients have been given gold therapy. Hartung noted only one death in well over a thousand cases. Hench reports that the average percentage of reactions by ten writers he reviewed was 37, ranging from 8 to 61.

Amongst our first 70 cases reported in 1937 we averaged 25% of complications. In a subsequent series of 183 cases, in many of which the patient had three or four courses, we had 33% of complications, almost three-quarters affecting the skin.

Freyberg noted the following incidence of toxic reactions in relation to dosage:

Dosage 100 milligrammes weekly, complications 41%.
Dosage 50 milligrammes weekly, complications 30%.
Dosage 25 milligrammes weekly, complications 18%.

The rheumatoid patients are more susceptible than those affected by ankylosing spondylitis, and in a series of 27 such patients treated before 1940 we observed only 15% of reactions. The rheumatoid group would seem to be much more susceptible than the tuberculous, as Blackburn in 1937 revealed very few reactions in 100 cases of pulmonary tuberculosis in which the dosage was approximately three times greater than in the series of Parr and Shipton. He reported that four patients had transient albuminuria, four had mild dermatitis, three had stomatitis—that is a total of 11%, in spite of the fact that his biggest dose was 0·4 gramme and the total dosage 0·86 gramme in six weeks.

In a group of 200 cases we could find no evidence of any relationship between complications and the sedimentation rate, but noted that those patients showing vasoconstrictive phenomena were more liable to albuminuria and blood dyscrasias. For this reason this particular variety is closely watched. As pointed out by Shipton and Parr, punctate basophilia is liable to develop very early and reach a high figure in patients who are susceptible to gold. As the hæmatological examination is tedious and time-consuming, it is impossible to carry out such counts to estimate punctate basophilia as a routine procedure. Even on small dosage, approximately one-third of arthritic patients will develop some complication, mostly concentrated in the skin, fortunately in most cases only mild. There is no known method to determine which patient will react badly, and all the supposed danger signals, such as low leucocyte count, eosinophilia, albuminuria and pruritus, are not always valuable. However, if the rule is followed to stop therapy when such signs are present, severe toxic reactions will be all the rarer. Until a non-toxic gold compound is made available, the price of prevention of serious complications will be low dosage and constant vigilance as regards the skin, urine and blood.

Gold therapy in rheumatoid arthritis cannot be undertaken lightly, and one has remarked: "anyone using the drug is at its mercy". The prevention of toxic reactions is not possible, but the reactions can be minimized. The vitamins, especially ascorbic acid, contrary to the state-

ments of many advocates, are not of any value. When reaction occurs the drug must be stopped.

Treatment of Toxic Reactions.

The treatment of toxic reactions has undergone a very great change since BAL (British anti-Lewisite) has been added to the therapeutic armamentarium. Its official name is 2,3-dimercapto propanol and it is a detoxicant of certain metals, reports from American, English and Australian sources pointing to its rapid efficacy. Exfoliative dermatitis, severe thrombocytopenic purpura and granulocytopenia have responded rapidly to BAL. However, one case of dermatitis unresponsive to the drug has been reported. Penicillin is of value in agranulocytosis resulting from chrysotherapy. The usual therapy of transfusions *et cetera* is valuable in complications affecting the hæmatopoietic system.

Relapses.

Relapses have been common in gold therapy, their frequency leading at times to pessimism regarding the effectiveness of chrysotherapy. Ragan and Tyson found in a follow-up of 142 patients that only 13% had not suffered relapse after three years, whilst 75% did suffer relapse and only 6% could be classified as having sustained five-year cures. Of those who did suffer relapse, they stated that 80% were improved by subsequent therapy. Most authorities give figures of 50% of patients improving with subsequent courses of gold. All authorities agree that the relapse rate is very high, but very few agree that 80% will subsequently improve with further chrysotherapy, and many patients, in spite of a repetition of courses, will steadily go down hill. It has been this failure of renewed treatment that has brought forth the crop of new compounds of bismuth and copper.

Results.

Most authorities are chary about figures of cure, and very few will venture past 10%. However, as regards improvement, nearly all give figures of appreximately 60% to 70%, which has led to some caustic remarks regarding the inevitable 70%. Ray in 1943, using calcium aurothiomalate in 50 cases, observed definite improvement in 50% of cases, slight improvement in 25% and no change in 25%.

It is difficult to judge what is meant by improvement, but suffice it to say that most authorities report the greatest success in early cases and the least in the older ones. We have noted the best results in the periarticular varieties with low sedimentation rate and the least improvement or failure in those in which vasoconstrictive phenomena predominate.

When cartilage destruction occurs gold therapy is useless. For an obstinate joint which has failed to recover whilst others in the same patient have, we have given intraarticular injections of gold (0·01 to 0·05 gramme of aqueous "Myocrisin") with benefit.

Accessory Therapy.

Tegner, who opposes the employment of gold therapy until the failure of all other methods, in an article on the treatment of rheumatoid arthritis, states: "the most important treatment is physiotherapy which includes general and local rest followed by mobilization of the joints". Many other authorities stress the same. Blood transfusions, intravenous injections of calcium gluconate, iron therapy, insulin and glucose may all be necessary before chrysotherapy can be safely or profitably employed. Orthopædic measures are important. Because of the frequent failure of gold therapy we have tried the sulphonamides in small dosage over long periods, and these have taken pride of place in our attack on the problem in the initial stages. If they fail or are only partially successful, the other methods with or without gold therapy constitute our line of attack.

Let us remind ourselves of the variability of the disease, its protean nature and the biochemical abnormalities, and there will be less of the complacency regarding the efficiency of any one remedy and more of the broader aspects of a therapeutic approach to this difficult and age-long disease.

Conclusion.

To sum up, gold therapy is a valuable therapeutic weapon in the treatment of rheumatoid arthritis, but it must be handled with care and discretion; it has suffered from the abundant enthusiasm of its early advocates; it is still shunned by a few; but it falls far short of being the ideal tool of the rheumatologist. The future calls for a destruction of its toxicity, the beneficent task of collaborating clinician and chemist.

HUMAN TOXOPLASMOSIS, WITH REPORT OF A CASE.[1]

By A. R. Edmonds,
Perth.

Human toxoplasmosis, with reports of two probable cases, has been adequately described by Robertson in The Medical Journal of Australia of September 28, 1946. Most of the clinical and experimental work has been done in the United States of America. Sabin has played a large part in bringing the clinicians' notice to the condition in his excellent summary in "Advances in Pediatrics", Volume I, 1942.

Briefly, human toxoplasmosis is apparently a rare disease, widespread through the world, and is no respecter of age, affecting the *fœtus in utero* and the adult, as well as children. The organism has been known to protozoologists since 1908, but infection in humans was first recognized by Castellani in 1914 and proven by Wolf, Cowan and Paige in 1937. Since then increasing numbers of cases are appearing in the literature.

The Parasite.

The parasite is a protozoon with a rather simple life cycle. It infests a large variety of mammals and occurs also in birds. It is apparently an obligatory intracellular parasite and has a wide tissue range, but apparently it grows only in fixed tissue cells. Apart from some success in growing the organism in minced surviving chick embryo, animal inoculation has been necessary for propagation. There appear to be many strains, whose properties vary with the host, but Sabin's work suggests that there is probably only one immunological species and that passage and adaptation through various hosts are responsible for the apparent variation in virulence and tissue affinities.

The organism is rather delicate and needs special conditions for survival outside the body.

Pathology.

The pathology of toxoplasmosis has been very well described by Sabin in the above-mentioned article, and he quotes freely from the work of Wolf, Cowen and Paige. There seem to be three chief factors at work:

1. Spread through the blood-stream, whatever the route of infection. In certain and very varied tissues, according, it seems, to strain and host, there occurs a migration through vessel walls, associated frequently with thrombosis of the affected vessel. This results in local necrosis in areas which are at the same time heavily invaded by the organism. With healing calcification can occur.
2. A specific granuloma, generally associated with an involved blood vessel.
3. An actual toxin production, which is most apparent in the brain of subjects who have died of diffuse encephalitis. Here the organisms are very scanty and a microscopically discernible destruction of nerve cells and some infiltration of inflammatory cells are the main lesions seen.

[1] Read at a meeting of the Section of Neurology and Psychiatry, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

Serology.

Recovery from an infection with toxoplasmosis results in some degree of immunity of undetermined duration. Neutralizing antibody can be demonstrated by inoculation of the skin of a rabbit with serial dilutions of a suitable strain of the organism in the serum of an actively infected host or of a patient who has recovered. A complement-fixing antibody has been demonstrated, but is not yet reliable enough for diagnosis in human infections. A report recently suggests that, both in the human and in the rhesus monkey, antibody may disappear from the blood in as short a time as seven weeks.

Human Infection.

Neither the source of the organism nor the route of infection in man is known. A possibly widespread animal reservoir and an insect vector are suspected but not established.

Sabin lists six types of infection in man:

1. "Hydrocephalus" *in utero*: a form of encephalomyelitis in the fœtus resulting in stillbirth, in which cerebral calcification has been recognized before delivery.
2. Encephalomyelitis in the newborn, or congenital toxoplasmosis, in which death occurs a few days or weeks after birth.
3. Congenital infection, in which the patient survives, showing in infancy or early childhood the tetrad of signs: (i) internal hydrocephalus, (ii) cerebral calcification, (iii) chorioretinitis and (iv) clinical signs of cerebral damage.
4. Acquired infection in early childhood, manifesting itself as atypical encephalitis, resulting in death or recovery with or without permanent damage to the central nervous system.
5. An acute fever in adults, associated with a skin rash and atypical pneumonia, which also may result in death or recovery.
6. Non-apparent infection, demonstrable only by the presence of antibody, or the production of an infant infected *in utero*.

Diagnosis.

Absolute diagnosis in a suspected case can be made only by cultivation of the organism in a number of laboratory animals by inoculation of material from the patient. Cerebro-spinal fluid, blood, bone marrow, muscle biopsy, sputum, or tissue or fluid obtained *post mortem* is used in appropriate cases.

Presumptive diagnosis can be made by demonstration of the presence of specific neutralizing antibody, and for this a supply of a virulent strain of the organism is necessary. A presumptive diagnosis is also justified in the presence of a typical clinical picture and microscopic demonstration of the organism. And finally it is justifiable to make a presumptive diagnosis in a case in which are presented the tetrad of signs listed in Sabin's fourth type of infection.

Report of a Case.

A baby girl, aged fifty days, was admitted to the Children's Hospital, Perth, on February 20, 1947. She was a first child, delivered by forceps; her weight at birth was six pounds five ounces. There was no evidence of birth injury observed. Her condition from birth to time of discharge from the maternity hospital was satisfactory. The mother had been well throughout the pregnancy. The child was breast fed for two weeks, was weaned for unstated reasons, and was fed subsequently on a dried-milk mixture. From the age of two weeks the baby took "fits". At some time after this signs of hydrocephalus were observed, the exact time being impossible to elicit. The child's weight had reached eight pounds three ounces, but was falling, being seven pounds seven ounces on her admission to hospital.

Examination at that time showed a rather wasted baby with gross hydrocephalus. Lumbar puncture one day later produces a very slow drip of amber-coloured fluid, the pressure being too low for accurate measurement. Compression of the jugular veins did not perceptibly increase the rate of drip. The fluid contained more than 200 milligrammes of protein per 100 millilitres, and there were 150 leucocytes per millilitre and a few red blood cells. Gram staining of the fluid showed degenerate polymorphonuclear cells and mesothelial cells; no organisms were recognized, and attempted culture of the fluid on blood-agar produced no growth.

Three days after the child's admission to hospital the subdural space was tapped on both sides in the fronto-parietal suture. A little bright blood and about one millilitre of cerebro-spinal fluid were obtained on each side. The next day a ventriculogram was performed by puncturing the right lateral ventricle and replacing cerebro-spinal fluid with air. Films obtained were of poor quality, but demonstrated gross distortion and enlargement of the lateral ventricles. The fluid obtained from the ventricular system was xanthochromic and under considerable pressure. There were 38 leucocytes per cubic millimetre, 800 milligrammes of protein per 100 millilitres and 580 milligrammes of chlorides per 100 millilitres; there was no growth on attempted culture.

From the time of admission the child was difficult to feed and had occasional mild twitchings. No therapy was applied. The condition deteriorated steadily and the child died on March 9, 1947, seventeen days after admission, at the age of sixty-seven days.

Post-Mortem Examination.

The general appearance was that of a hydrocephalic child with an emaciated body. The lungs and pleura were macroscopically normal; no glandular enlargement was present in the thorax. The heart was small, but otherwise normal. The liver was rather congested. The spleen was enlarged to about twice the normal size. The kidneys were normal except for a double ureter on the left side. The stomach and intestines were normal; there was an unusual and marked absence of enlargement in the lymphatic glands commonly seen at this age.

Cranial Cavity.—The cranial sutures were very widely open and the bones thin enough to cut readily with scissors. The brain was grossly distended, the gyri were flattened and the sulci were obliterated in the manner usual in hydrocephalus. The surface of the cerebrum was a very pale grey. Scattered over the surface were areas of unusual appearance. These were irregular gyrate to circular figures, varying in size, having a yellow centre and a whitish granular edge. When the brain was removed there was found to be gross involvement of the base in a plastic meningitis, the normally seen structures, such as the optic chiasma *et cetera*, being practically unrecognizable. The meninges over the vault were not greatly involved, there being one area, however, in the right parietal region where the brain was adherent to the parietal bone. Below the *tentorium cerebelli* there was a marked contrast; the contents of the posterior fossa appeared almost normal. The consistency of the cerebrum was that of a not very thick, smooth porridge, and it broke up and almost flowed around the table when handled. The ventricles were grossly distended, no chorioid plexuses were to be seen, and the ependyma was thickly covered with a fibrinous exudate which had a very close similarity to the bread-and-butter appearance of rheumatic pericarditis. The ventricular fluid was glairy and yellow. There were several areas of firm consistency throughout the cerebrum, and in one occipital pole a rather hard lump. Pieces of the brain were preserved for examination, some fixed in formol saline, some collected as aseptically as possible. Some of the intraventricular fluid was also saved. The opened skull was photographed before the brain was touched.

Histological Examination:—Immediate examination of the ventricular fluid was carried out. A number of the ordinary stains were tried, and Leishman's proved by far the best. It demonstrated the typical morphology very adequately. All these smears, however, faded very rapidly. The next day, after rather inadequate storing, some of the tissue was injected intracerebrally into a rabbit, but no lesions resulted. Dr. Kingsbury, of the Public Health Laboratory, kindly carried out this part of the investigation, and assisted us in the section examination.

For the following description, I am indebted to Dr. V. A. Fergusson Stewart:

Brain Smear (Leishman stain). Small yeast-like bodies with red chromatin network present. Appearances consistent with toxoplasmosis.

Brain. Gross necrosis and breakdown of nervous tissue, multiple protozoa present, some in pseudo-cyst form.

Liver. N.A.D. except congestion.

Spleen. Increase in fibrous and connective tissues; cells all of epitheloid type, with gross eosinophilia. No protozoa seen.

AORTITIS CAUSED BY CONGENITAL SYPHILIS.

By Charles Engel, M.D. (Budapest),
Sydney.

Earlier authors were of the opinion that congenital syphilis did not produce aortitis (Lanceraux, 1873). From the historical standpoint it is worth mentioning here that the great Hunter, in the late eighteenth century, held the same view concerning acquired syphilis. Till the end of the nineteenth century only a few reports had been published about alterations of the aorta in cases of congenital syphilis. When at the beginning of this century interest in syphilitic aortitis arose and rapidly widened all over the globe, it could soon be stated that aortitis caused by congenital syphilis occurs incomparably less often than in the case of acquired syphilis; that, as a matter of fact, it is a very unusual disease. It is now common knowledge that the specific lesions of the aorta start very shortly after the acquiring of the syphilis, but the first symptoms do not become manifest till after a span of fifteen to twenty years. In the young person, therefore, the first symptoms should appear at the age of fifteen to twenty years. Such cases were indeed observed and published, but in a number quite insignificant, indeed trifling, compared with the hundreds of thousands of cases of aortitis caused by acquired syphilis.

What may be the reason for this striking observation? It is well recognized that in the infected embryo the spirochætes spread everywhere and invade every organ, in the same manner as is known for the "stage of generalization" after syphilis has been acquired. Spirochætes settle therefore even in the wall of the aorta, infection being more massive, in greater amount, than in the adult. It is also well known that the spirochætes produce there histological changes too. Already, in 1905, Wiesner had demonstrated pathological changes in the aorta of syphilitic infants born dead or living for three months. These were: lymphocytic infiltrations in the media, partial or total occlusions in the *vasa vasorum* and proliferation in the adjacent tissue. Bruhns corroborated these findings. Though subsequent investigations, especially those of Scharpff and Werlich, raised doubts that these changes were of syphilitic origin, we can regard it as proven by the continued studies of Wiesner, Rach and Wiesner, Rebaudi, Thoenes *et alii* that in the aorta of infants with congenital syphilis the same changes may be revealed as are present in the first stage of aortitis caused by acquired syphilis.

About the frequency of this occurrence little has been said; Rach and Wiesner estimate it at 64·7%. It can be regarded, however, as certain that from this initial condition the typical picture of progressive aortitis develops much less often than in the aorta of syphilitic adults. Detailed further investigations by a large number of authors, such as Kerl, and especially the North American workers Previtalli *et alii*, and MacCulloch *et alii* have confirmed this statement. With my collaborators in the Policlinic in Budapest I studied our patients with congenital syphilis for twenty years, but no case of aortitis was revealed. During this time I interviewed a large number of well-known pædiatrists with wide experience in congenital syphilis; none of them ever made a diagnosis of aortitis in his patients with congenital syphilis, "in spite of special and utmost care in investigating them so in early childhood as in the first and second decade".

The occasional publications in the literature deal mostly with one or two cases, seldom with more. Schlesinger, who was very interested in this condition and had a very rich source of material for a remarkably long time, has recorded four cases. In Australia I am aware of two cases; obviously more could be detected. One has been reported in this journal by H. Patterson in 1942. In the case of the second I have to thank C. Duncan, of Hobart, for his personal communication; he found syphilitic aortitis at the necropsy on a fifteen-year-old boy with paresis of the insane. The young patients described in the literature displayed mostly symptoms of progressive heart failure or suffered from pains like those of *angina pectoris*. Some died suddenly on exertion, and the post-mortem investigation revealed in some cases aneurysm or even its rupture, as in Patterson's case.

On the basis of these collected data I have to draw the conclusion that progressive aortitis caused by congenital syphilis is an extraordinarily rare disease. I do not find justified the contrary conclusion of some South American authors, namely, that evidence of aortitis in children with congenital syphilis is not so unusual. Beretervide and Beretervide diagnose aortitis in children if they find the aorta broader on fluoroscopy than would correspond to their age. Gesteira diagnoses aortitis on finding the second sound of the aorta abnormally loud and "tambour-like". These authors assume that the intensity of the abnormalities diminishes distinctly after antisyphilitic treatment. As the pathological corroboration of their findings is missing and as they have not been confirmed since their publication, I would hardly regard their conclusions as convincing.

What is the reason that aortitis develops so seldom and progresses even less often in congenital syphilis? Stadler in his book gives the opinion that aortitis is so rare because the patients die in a relatively early period of life. This supposition does not satisfy me; cirrhosis of the liver, tabes, paresis of the insane *et cetera* are not so rare in an age when the phenomena of progressive aortitis should already be evident. One might suppose, of course, that in the youth of fifteen to twenty years of age aortitis might be present yet not exhibit any symptoms because it is of the abortive type (Engel), or because at this age there are not yet present those alterations of the aorta, fibrosis and sclerosis, which promote the manifestation of symptoms in adults fifteen to twenty years after the infection. This supposition is untenable too, because, as already mentioned, patients who die at this age are found to have aortitis only very exceptionally.

At present this problem is not solved.

It seems that it is a special constitutional feature of early childhood that the spirochætes are controlled so easily in the aorta wall. Perhaps the antisyphilitic treatment is of special effectiveness in early childhood, at least against the changes in the aorta, though it seems that the initial alterations in the media of the aorta are controlled without specific treatment also. What is the essential mechanism of this successful defence of the young organism is still completely unknown, as also hardly anything is known about the problem of the tropism of the spirochætes for the aorta and central nervous system. One has to take into consideration the fact that throughout the whole vascular system the dynamic load is the greatest just in that part of the aorta where aortitis is the most frequent. I have to mention also that in contrast to the location in the aorta, an analogous change in the pulmonary artery is very rare, still rarer than in the aorta in congenital syphilis. It may be that the lower burden in the pulmonary artery is of decisive significance in this respect. If that is so, this might have some bearing on the aorta in childhood too. Of course, the environmental factors (stress, hard labour, worry, alcohol, nicotine) should be regarded as very important in promoting aortitis; these explain why aortitis is so much more frequent in adult males than in females, the ratio being between 5:1 and 6:1.

I believe it is worth mentioning that aortitis due to congenital syphilis may appear at the same age as that caused by acquired syphilis, that is to say, in the thirties or forties. Some examples of this kind are described in the literature. From my private practice I have a record of two such cases. In a woman of twenty-nine years with scars on the cornea caused by keratitis and bone scars as a result of congenital syphilis, I found a well-marked dilatation of the *aorta ascendens* with an amphoric aortic second sound. She complained about retrosternal pains on exertion. The Wassermann reaction was present ("+++"). Antisyphilitic treatment controlled the pains completely. The second case was that of a woman of forty-two years, whose father I had treated many years before for tabes and aortic incompetency. She called on me

because of very severe headaches. Three younger sisters suffered from congenital syphilis; the patient herself had had several courses of antisyphilitic treatment in early childhood. Her aorta was well dilated; there was a systolic murmur audible over it with a strongly accentuated second sound. The blood pressure was normal. The Wassermann reaction was present in her blood, but absent in that of her husband. The patient certainly never showed any signs of acquired syphilis. Antisyphilitic treatment checked the headaches in a short time.

In such cases I believe we have to suppose that the harboured infection, or rather a latent change in the aorta wall, has been activated by the stress of adult life, while manifestations of the condition have been promoted by changes in the aorta due to age.

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SOME OBSERVATIONS ON RECENT ADVANCES IN THORACIC SURGERY.

By H. D'ARCY SUTHERLAND, M.S., F.R.C.S., F.R.A.C.S.,
Adelaide.

I AM sure you will appreciate my difficulty in attempting to present some points of interest from a specialized branch of medicine to an audience of such diverse leanings.[1] It is impossible for me to touch on all the aspects of thoracic work with which I have been in contact, so I will confine my remarks mainly to some principles of collapse therapy in tuberculosis and some aspects of the early diagnosis of carcinoma of the lung.

In the last decade penicillin has lowered mortality and morbidity to such an extent that the thoracic field of surgical endeavour has been widened considerably. Thus penicillin and surgical courage challenged more and more the clinical acumen of the physicians, so that a parallel advance of thoracic medicine and surgery resulted. In a thoracic unit there is no arbitrary division of cases into medical and surgical, because the work overlaps too much for this, but all work is carried out on a team basis. Physicians and surgeons do rounds together; added to this, the radiologist, physiotherapist and anæsthetist are consulted on most cases; certainly on all cases involving operation. I shall always remember a statement made by one of the surgeons at a meeting of the Thoracic Society in Birmingham this year: "He is a poor physician who does not ask the cooperation of a surgeon even in the early stages of a disease, and he is an imperfect surgeon who does not ask for the help of a physician."

I found working under these conditions a great pleasure and, what is more important, modern standards of treatment of chest diseases cannot be attained without such a background of team work. Weighty decisions have to be made and it is only fair to the patient to give him the best advice available. In England it is estimated that one thoracic surgical unit is required for each million of the population. In Australia I think that with the high standard of general practice and with it a higher standard of medical education of the public, we could have three units in Sydney, two in Melbourne, and one each in the other capital cities. It is not economical in staff and equipment to separate the tuberculosis and non-tuberculosis work; for this reason in England it has been found practical to set up thoracic units in the bigger sanatoria, as well as in association with the big general hospitals. Sanatoria of sufficiently high standard do not exist in Australia; hence the latter course is the only one open to us, and this in fact is what is being done, roughly in the distribution I have stated.

It was my good fortune to have been associated with Harefield Hospital for the major part of my stay in England. The hospital, as it now stands, is in the same grounds as the general hospital of the Australian Imperial Force in the 1914-1918 war. After World War I the property was bought by the Middlesex County Council and the huts were used as a sanatorium. The modern buildings which have replaced the old huts were started in 1931, but the work was not completed until about 1937. During the recent war the hospital continued as a sanatorium in a restricted way, but its major function was as a thoracic surgical unit (Emergency Medical Services) and as the sector hospital for Saint Mary's, Paddington. The thoracic unit has continued to flourish in these surroundings and is now established as one of the main chest centres in England. The beds previously occupied by Saint Mary's are now available for sanatorium patients. The work is directed by the consultants, who also visit other sanatoria and hospitals, but the bulk of the routine work is done by the full-time staff.

In England before the advent of national medicine the standard of treatment of tuberculosis was in direct proportion to the financial state of the county concerned. Harefield, being in the predominantly "middle class" county of Middlesex, is one of the best sanatoria in England. Theoretically the only patients admitted to sanatoria are those suitable for active treatment; that is to say, no beds are filled by "hopeless" patients while treatable patients are "waiting". The patients are called in from a central waiting list and whilst awaiting admission they are treated by the physicians in the chest dispensaries or in special wards of the general hospitals. The non-tuberculosis patients nearly all come from other hospitals, but a few are referred from the chest dispensaries and from general practitioners. The chest dispensaries thus provide some of the earliest carcinomata; because besides treating domiciliary and ambulant tuberculosis patients they serve a very useful purpose as preliminary diagnostic centres for all chest diseases.

Patients responding to treatment, on discharge from the sanatoria, go back to the chest dispensaries for follow-up care.

It is impossible even to outline the modern management of tuberculosis in so short a time. Instead I will refer to a few of the major principles which are now accepted as the basis of sound therapy. In passing I do not want you to be mistaken, in that by my frequent reference to

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 26, 1948.

artificial pneumothorax I have taken on the mantle of a physician. This measure is entirely in the physician's hands and should remain there; but so much of the success or otherwise of collapse therapy is determined in the first months of treatment that I want to stress the importance of this measure's being used effectively and at the correct time and, if unsuccessful, being abandoned for some other collapse measure before irreparable harm is done. The long-term management is essentially the responsibility of physicians; however, the surgeon is often consulted early, whether immediate surgical treatment is envisaged or not.

On admission to the sanatorium the patient and his disease are carefully assessed, then, if collapse measures are indicated, the most suitable type is decided upon. The indications are nearly always present for artificial pneumothorax to be tried first, but by no means in all cases. This brings me to one major fact that I should like you to take away with you this evening, namely, that major collapse measures should never be undertaken in acute exudative lesions or in recent exacerbations of this type on top of chronic disease. When I say "major collapse measures" I most certainly include artificial pneumothorax in the group. If exudative disease is present, a preliminary period of bed rest is essential, possibly assisted by streptomycin, pneumoperitoneum or phrenic crush, depending on the indications, the appropriate collapse measure being initiated only when some X-ray and clinical evidence of healing is apparent.

If artificial pneumothorax is decided upon, induction is performed and the case is reassessed critically immediately after induction. We may be faced with one of several situations—failure to obtain a pleural space, gross adhesions, adhesions suitable for division or a good artificial pneumothorax, the chance of the last-mentioned being as low as 10% according to most authorities. Unless the artificial pneumothorax is perfect, thoracoscopy should be performed early, because it is both a diagnostic and a therapeutic measure, with practically no risk attached to the measure *per se* in good hands. Whilst I was at Harefield some 400 thoracoscopies were performed with no serious complications. There is no more certain method of causing a tuberculous empyema than by allowing a tuberculous lung to be suspended in adhesions. Therefore, unless there are extenuating circumstances, if the artificial pneumothorax is not, or cannot be rendered efficient, it must be given up at this stage and not some months later when some irreparable damage, such as a tuberculous empyema, has resulted. Artificial pneumothorax is, I think, one of the best examples of the old surgical axiom: "The results of any therapeutic procedure depend on the way it is used and not the fact that it is used." Rafferty in his book on artificial pneumothorax has stated the position very clearly:

> The first few months of an A.P. are the most important because it is during this time that most of the vital decisions are made. The need for collapse therapy must have been established and the choice of a reversible measure justified. Effectiveness must be assessed and the question of adhesion-section considered and if required it must be skilfully performed at the correct time. If evidence of mistakes with A.P. became apparent more quickly, their connection with complications would be better understood and the need for careful management more obvious. The complications of A.P. which were very frequent in the early days of its use we now know are nearly all avoidable if the collapse program is properly planned and A.P. only used and maintained when the indications are there and not just as a first inevitable step in a series of ill-judged blunders.

If artificial pneumothorax fails or the subject is not suitable for this measure, then the possibility of other procedures, such as extrapleural pneumothorax, thoracoplasty or resection, has to be considered, the fact being remembered always that any such treatment is only a local measure for a general disease. Pre-operative and post-operative physiotherapy plays a very big part in the management of patients undergoing surgical collapse operations. Before operation the patient is taught how to posture himself, how to perform arm exercises he will be using after operation and the necessary breathing exercises. After operation active measures are instituted to prevent collapse in normal lung tissue, as well as exercises to encourage arm movements and the maintenance of good posture. Last year Mr. T. Holmes Sellors analysed some 600 personal cases of thoracoplasty and found that he had a 3% immediate mortality, 90% cavity closure and 80% sputum conversion, and that 80% of the patients were doing some form of work.

The difficulty with the surgery of lung tuberculosis is not so much the technical problem, difficult as it can be on occasions, but rather the selection and timing of the methods available to combat the disease. A cavity is always a potential danger and must be dealt with if possible. I have often heard it said that "of patients with untreated cavitation only 20% survive five years, while of those successfully treated less than 20% are dead in five years". Rest is probably still our best weapon, but with our minds always on function, after the initial period of observation and assessment and when a line of treatment has been undertaken, then the patient is always graded up in exercise to the estimated limit of his tolerance. Regular critical reassessments of the patient and his disease are made, the old method in which some arbitrary length of bed rest was decided upon at the outset of treatment being thereby replaced.

In the last decade a more rational therapeutic programme has been evolved for one or more of the following reasons (after Rafferty): (i) improved sanatorium facilities; (ii) improved knowledge of the formation and behaviour of cavities; (iii) knowledge of the course and implication of endobronchial tuberculosis; (iv) realization, through observation and analysis of the late results, of the long-term importance of a careful choice of cases for the various collapse measures; (v) increased use of and improved technique of adhesion section; (vi) improvement in the application of thoracoplasty.

Carcinoma of the Lung.

I should like to add my voice to the loud and continuous plea for the early diagnosis of carcinomata in general. In carcinoma of the lung the onus is clearly on the clinician, because the patient in the operable phase often suffers only very minor symptoms; likewise the signs may be there but appreciated only by careful analysis. The lung is one part of the body where early diagnosis is often possible if it is only considered.

For practical purposes all carcinomata of the lung arise in the bronchial mucosa. The cellular pattern, however, varies considerably, as does the natural history of the disease, depending, of course, on the degree of undifferentiation of the cellular structure. The tumour affects the lumen of the bronchus and herein lies the secret of early diagnosis. The symptoms and signs result from the actual location of the growth relative to the bronchial tree *plus* the likelihood of surface or central necrosis of the tumour itself. If the disease is in a major bronchus, then a lobe or a whole lung may collapse; if in a minor bronchus, segmental collapse alone may result. When the carcinoma arises in a terminal bronchus, then the larger bronchi are only involved secondarily; hence in such cases we have to rely on other means of diagnosis. As the tumour affects the lumen, cough and hæmoptysis may arise even before there is a demonstrable area of collapse. When a segment does collapse, it becomes infected invariably and signs of localized pneumonia result, which, because of the underlying cause, will not clear up with the usual treatment. From what I have just said we get our first clues to early diagnosis, namely, cough, hæmoptysis, "pneumonia" (not responding to usual remedies) and that new physical sign of this modern age—X-ray "pick-up". Most other symptoms and signs are those of extension of the disease and usually of inoperability. Under such a heading I would include pain (other than pleural pain from "pneumonia"), cyanosis, anæmia, dyspnœa, clubbing, loss of weight, voice changes, palpitation and "Pancoast's syndrome".[1]

[1] Dr. Sutherland then showed a series of slides and X-ray films of patients who had presented themselves with one or other of these symptoms.

The diagnosis of carcinoma of the lung may be absolute by bronchoscopy and bronchial biopsy when accessible, or reliance may have to be placed on indirect methods, such as the demonstration of bronchial block by bronchogram, and other circumstantial evidence, together with the exclusion of other possibilities by a process of elimination. Bronchoscopy, apart from the actual seeing of the tumour and the taking of a biopsy specimen, provides important evidence in the assessment of operability. Other investigation includes a thorough clinical and X-ray examination (including "straight" films, tomography, bronchograms and screening). The sputum is examined to exclude tuberculosis, and in certain circumstances a search for carcinoma cells may be made.

Full assessment is too involved to discuss here tonight, but let me dismiss it shortly by saying that, when conditions are suitable, the only treatment giving any hope of cure is operation. This usually means a pneumonectomy and less commonly a lobectomy.

Pre-operative, operative and post-operative management is also beyond the scope of this talk. The pre-operative treatment and post-operative treatment are both directed towards early return of function of the chest and the patient. Penicillin has made modern chest surgery possible by eliminating the erstwhile 100% risk of empyema in the pleura—this being of particular importance in penumonectomy, after which a dead space exists for a time at least.[1]

Operative technique is complicated and can be carried out successfully only on a team basis. In this type of surgery the unexpected may happen quickly, hence it is essential that everyone involved in the operation must know what the other members of the team are doing at all times. In passing I should like to quote the operative figures for Harefield Hospital for the time I was there: thoracotomies, 166; lobectomies, 70 (no deaths); pneumonectomies, 58 (five deaths); others (various), 38 (four deaths). This series, with a "hospital mortality" of 5·1%, represents operations for all diseases, but at the same time gives an indication as to the mortality figures which can be expected in a properly organized unit.

Cardiac Surgery.

The surgery of the tetralogy of Fallot and coarctation of the aorta was just being developed while I was in England and, apart from Brock at Guy's Hospital, the other chest centres were all dealing with their first few cases. I saw Blalock's film and heard him speak on several occasions while he was in England, but getting in to see him perform an operation was beyond my powers of diplomacy. I received a letter only a few days ago from a friend of mine, who now has his own thoracic surgical unit in England, telling me of his first Blalock operation. It cheered me considerably to hear from him that his patient was still alive and that it was not nearly so difficult as he had imagined. The surgical treatment of patent *ductus arteriosus* and to a lesser extent of constrictive pericarditis was more commonplace, and I have seen some excellent results in both of these conditions. We had two patients with constrictive pericarditis in the last few months, and the sudden immediate improvement was quite spectacular. In both cases ascites had been present for five and three years respectively, and neither patient required paracentesis after operation.

Surgery of the Œsophagus and Stomach.

The surgery involving the transthoracic approach of the œsophagus and stomach is divided between the thoracic surgeons and some general surgeons who have interested themselves in this work. The surgical repair of congenital strictures of the œsophagus is now meeting with a measure of success. This is technically difficult for both the surgeon and the anæsthetist and can succeed only if the patients come to operation in the first few hours after birth and before too much food, mucus *et cetera* have found their way into the lungs through the tracheo-œsophageal fistula or through aspiration of regurgitated œsophageal contents. Lipiodol and barium administered by the well-meaning clinician will have the same effect. The surgery of this region in adults mainly centres around carcinoma of the œsophagus and the various types of diaphragmatic hernia. We operated on about ten patients suffering from the former with only one hospital death. Under ideal conditions mortality and morbidity are reduced to such an extent that one is justified in recommending operation in all cases in which it is technically possible. There is a tendency in some clinics to carry out this procedure as a combined operation between a general and a thoracic surgeon working as a team and thereby sharing the strain of what is often a four-hour or five-hour undertaking. It is not practicable for the two to work at once, as in combined abdomino-perineal excision of the rectum, but each in turn assists the other to perform his part of the operation. The essential step in the operation is to free enough stomach and/or small bowel below the growth to allow the carcinoma to be removed in block and to permit of anastomosis of the two ends. The trouble about this operation when carried out by one surgeon is that the most exacting part of the technique, namely, the anastomosis (which is more difficult than the usual abdominal anastomosis for many reasons), comes towards the end of the long struggle.

Once again early diagnosis is essential if any good results are to be attained. With any patient over the age of forty years complaining of dysphagia the onus is on the medical attendant to disprove the possibility of carcinoma as early as possible and not to allow him to drift on to inoperability. Absolute diagnosis can usually be established by œsophagoscopy after one's suspicions have been roused by the history and barium-swallow examination. L. G. Blair, a radiologist who has had considerable experience in these matters, used to say that barium-swallow examination alone was not sufficient evidence on which to exclude carcinoma if reasonable suspicion had been aroused.

Bronchiectasis.

The surgical treatment of bronchiectasis forms a big part of chest surgery in England. Many patients passed through our hands, and I am pleased to be able to say that not one died as a result of our efforts. The problem of surgical treatment of bronchiectasis is not at all an easy one. There is no question of operation's being a cure-all for the disease, because in many cases the lesions are too disseminated to warrant anything but conservative treatment. Careful preliminary observation and assessment have to be completed before a decision is reached. When the subjects are correctly chosen for operation the results are very gratifying, because the patient's outlook on life is completely changed. Operation is indicated in some bilateral cases, particularly in children, but the best results are in cases in which the disease is confined to one side and preferably one lobe. In the latter type the diseased lobe is often just a collapsed mass of lung tissue containing bronchiectatic cavities. The bulk of the lobe is small relative to the remainder of the lung; hence no compensatory adjustment is required, convalescence being thereby made rapid and complete. Physiotherapy, important as it is in all chest surgical work, probably plays its biggest part in all aspects of the management of bronchiectasis.

Conclusion.

In closing I should like to express before the members of this Branch my gratitude to the trustees of the Nuffield Foundation for their part in assisting me to go to England and for the grand opportunities they afforded me in my work there; and to Mr. T. Holmes Sellors and to the staff of Harefield Hospital in general for their teaching, encouragement and friendship during the very happy months I spent in their midst. Finally I should like to express to you, the President, and to the members of the Branch, my deep appreciation of the honour you have done me in asking me to address you this evening.

[1] Dr. Sutherland then showed a further series of slides of X-ray films showing some of the pathological states which could be confused with carcinoma of the lung. He made special reference to bronchial adenoma, lung abscess, tuberculosis, tuberculoma, empyema, paratracheal cysts, hydatid disease, neurofibroma and retrosternal goitre.

Reports of Cases.

DACRYOCYSTO-RHINOSTOMY IN AN INFANT

By K. B. Redmond,
Orange.

Congenital blockage of the naso-lachrymal duct is of comparatively frequent occurrence,[1][2] but the number of cases not responding to conservative measures, such as the use of astringent guttæ and sac pressure or to syringing or probing under general anæsthesia, is exceedingly small.

The naso-lachrymal duct develops along the line of the cleft between the lateral nasal and the maxillary processes. The maxillary process overlaps the lateral nasal process and tucks in a fold of ectoderm; this forms a solid rod of cells, which is later canalized, forming the duct.

The last part to open is the inferior orifice, where the duct opens into the inferior meatus of the nose, and this usually becomes patent at the end of the eighth month.[3]

Congenital lachrymal obstruction, evidenced by a watery eye and a muco-purulent discharge when the static contents of the sac become infected, is therefore usually due to a plug of epithelium low down in the duct.

Pressure exerted over the lachrymal sac with the ball of the thumb, and directed in the line of the duct, will drive a column of fluid down the duct, and repeated efforts may finally dislodge the plug and allow of free natural drainage. If this does not occur, the passage of a number 1 and a number 2 Bowman probe under general anæsthesia will usually dislodge the plug, and sac pressure will then keep the canal open. Occasionally probing will need to be repeated.

Cases in which the duct is permanently and solidly blocked and a probe cannot be passed are due to trauma, nasal operations or infection.

The following case is one in which excessive fibrous-tissue formation at the junction of the sac and duct, due to repeated lachrymal abscesses, resulted in a solid obstruction; the condition was cured, with good functional result, by a dacryocysto-rhinostomy.

It is of particular interest, for this operation is not commonly performed or indicated in cases of infants. In a report in 1946[4] of 50 patients who underwent dacryocysto-rhinostomy there are no cases among infants and, indeed, the writers say: "We have had no experience of the treatment of naso-lacrimal obstruction in children, except by probing in infantile cases, and we are not able to assess the place held by dacryocysto-rhinostomy in its treatment."

Clinical Record.

E.T., at the age of three weeks, developed a left lachrymal abscess, which was opened. When he was aged six weeks it was again curetted and packed with sulphanilamide powder. Two months later (he was then aged three and a half months) an abscess again formed and it was widely opened, carbolized and packed with gauze. Three weeks later this procedure was repeated. During the next twenty-six months the eye remained watery, but no abscesses or mucocele formed. However, at the end of this time a large infected mucocele was present, which regurgitated copious muco-purulent material on sac pressure.

At the age of three years he was admitted to hospital with a large left mucocele, which contained about one millilitre of muco-purulent material. This was syringed out and lipiodol injected; radiography then revealed a large lachrymal sac, completely blocked at its lower extremity.

In view of the presence of the large sac it was decided to perform a dacryocysto-rhinostomy. It is of interest to note that this sac either had not been destroyed by the repeated incisions, packings and carbolizations or had regenerated from some remaining epithelial tissues.

Under general anæsthesia an incision was made, commencing midway between the inner canthus and the root of the nose and carried down the side of the nose, curving slightly outwards, so as to end about three-quarters of an inch vertically below the lower punctum (it was thus on the nasal side of the scars of previous drainage operations). The incision was carried right down to the bone through the soft tissues, and all the tissues, including the periosteum and the attachment of the internal lateral ligament, were displaced outwards. The lachrymal sac was seen lying deeply in the structures so everted. The lachrymal fossa was exposed and the sac dissected outwards from it. With a gouge and hammer and nibbling forceps the floor of the fossa was removed and the nasal mucosa exposed. Flap incisions were made in the nasal mucosa and the medial wall of the lachrymal sac and the posterior and anterior flaps respectively brought into apposition. Stitches to unite these were not used.[5] The skin was closed with interrupted silk sutures.

On the third day the lachrymal passage was syringed and fluid passed readily into the nose and down the throat. This was repeated on the sixth, tenth, fourteenth and twenty-first days, and once or twice irregularly since to demonstrate patency.

At the present time, fourteen months after operation, the eye does not water and the sac has had no further obstruction or infection. The scar is negligible.

Acknowledgements.

I am grateful to Dr. A. E. and Dr. C. S. Colvin, Dr. R. S. Candlish and Dr. T. C. Meurer for their advice and assistance in the conduct of this case.

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(2) K. B. Redmond: "A Preliminary Survey of Ophthalmic Diseases in Western New South Wales", *Transactions of the Ophthalmological Society of Australia (British Medical Association*, Volume VII, 1947, page 126.
(3) S. Duke-Elder: "Text Book of Ophthalmology", Volume I, page 365.
(4) T. Keith Lyle, A. G. Cross, J. F. Simpson and G. A. Fraser: "Dacryo-cysto-rhinostomy", *The British Journal of Ophthalmology*, Volume XXX, 1946, page 102.
(5) T. Tyrrell: Personal communication.

Reviews.

HYGIENE AND PUBLIC HEALTH IN INDIA.

The appearance of a twelfth edition of Dr. Birendra Nath Ghosh's well-known "Treatise on Hygiene and Public Health" is not unexpected.[1] The author has ever pursued the course of keeping this work up to date. It was rendered almost inevitable, however, by the publication of the Report of the Health Survey and Development Committee in 1944. India, like other countries in the British Commonwealth, is planning for the future, and the programmes, both long term and short term, envisaged by the committee will have a profound effect on the health of her people.

Dr. Ghosh, in his interesting introductory chapter which traces the development of public health administration in England as well as in India, links these plans for the future with present practice. That there is scope for far-reaching measures is shown throughout the book. The table showing the mortality rates and the expectation of life at birth in India and certain other countries indicates that the health of India as a whole is at a low level. The results of investigation of the health of about 28,000 students in Calcutta show a high percentage of malnutrition (33%), of tuberculosis (23%) and defective vision (31%), but on the other hand reveal a surprisingly low percentage (19%) of dental caries, although the author regards it as high. He would be very shocked at the rate (95%) in Australian children.

Other indications of the low standard of hygiene are given in the article on houses and buildings, in references to the prevalence of "back to back" houses and in such comments

[1] "A Treatise on Hygiene and Public Health: With Special Reference to the Tropics", by Birendra Nath Ghosh, M.B.E., F.R.F.P. and S. (Glasgow), F.R.S. (Edinburgh); Twelfth Edition; 1948. Calcutta: Scientific Publishing Company. 8½" × 5", pp. 784, with illustrations. Price: 15s.

as: "In India it is rather exceptional to find a bakehouse built on sanitary principle. The law relating to them being very defective, any room or hut may be converted into a bakehouse", and in the description of the slum areas known as Bustees.

The treatise is obviously written to meet the needs of medical practitioners, health officers and students in India itself, and where it deals with the general principles of hygiene it differs but little from other text-books on the subject. In matters of detail, however, it is very different. The chapters on diet and foods deal not only with the dietary problems presented in a country inhabited by different peoples of different nationalities, castes and creeds whose food habits differ markedly, but with many foodstuffs whose names are unfamiliar to Australian readers. The epidemiological problems presented by large aggregations of people, often under very primitive conditions, in connexion with religious festivals and pilgrimages are not met with by Australian health officers, nor are the sanitary problems posed by the various methods of disposal of the dead. These points of difference, while perhaps robbing the work of some of its usefulness to Australian students, certainly add greatly to its interest in other ways.

The present edition is somewhat larger than its immediate predecessor. While the author found it necessary to delete certain portions, much new material has been introduced in almost every chapter. As in previous editions, contributions have been made by noted authorities on special subjects. The volume contains 764 pages with 153 illustrations and diagrams. Although the author ends his preface with a reference to abnormal conditions and the difficulty of getting paper and other materials and craves the indulgence of his readers for many shortcomings, there is little need for his plea, for the volume is well printed, the type is clear, misprints are few and the indexing is good. Although from the very nature of the work it is not altogether suitable as a text-book for students in temperate climates, it is a valuable work of reference to health officers in tropical regions.

EAR, NOSE AND THROAT DISEASES.

THE excellent reception accorded the first edition of "Diseases of the Ear, Nose and Throat" published in Australia has encouraged its author, Douglas G. Carruthers, to increase its circulation by arranging for the publication of the second edition in England.[1]

The book has been revised and brought completely up to date by inclusion in treatment, whenever applicable, of antibiotic chemotherapy. Additions to the text include a new section dealing adequately with tropical *otitis externa* and also one on local anæsthesia in dissection of tonsils together with a detailed and well-illustrated description of the operation itself.

As the treatise is essentially for the student and general practitioner but little is said of the operation of fenestration for otosclerosis and that guardedly. On the other hand, all the minor surgical procedures and examination methods are very clearly described and illustrated. The work is intensely practical and admirably suited to those for whom it is intended.

PUBLIC HEALTH.

"PUBLIC HEALTH ADMINISTRATION IN THE UNITED STATES", by Wilson G. Smellie,[2] is a work which should grace every Public Health Department library. Despite its title it is not a book dealing with bureaucrats and their departmental activities, but is an epidemiological approach to preventive medicine written for the guidance of medical students in the United States, but suitable for the public health administrator in any State in Australia. The subject matter is set out concisely and the points made are easily seen and assimilated.

Part I sets out the numerous functions of a public health service. Generally speaking, these are part of the activities of the State Health Departments of this country. These functions are related to the people as a whole, not to the individual.

Part II deals with the control of communicable diseases. The epidemiological considerations that affect control are first discussed and the preventive measures as regards the individual and the community, based on this epidemiology, follow. While the subject matter may be found in a number of text-books on preventive medicine, it is not set out as clearly, nor is the approach as practical as that of the author of this volume. Special emphasis is placed on seeking the cooperation of the community through health education and subject matter which can be used in educational work is given under each disease where applicable.

Part III covers what should be the various activities of a health organization, and is essentially administrative. The chapter on public health education is worthy of mention. It briefly covers all aspects of health education and emphasizes the pitfalls. The diseases of adult life are discussed from the administrative and epidemiological angles in the chapter on adult hygiene.

Part IV sets out the functions of and the relationship between the municipal, State and Federal Health Departments in the United States and is not applicable to this country.

Throughout the book the author expresses the opinion that "the prevention of disease and promotion of the health of the individual are essentially the functions of the practitioner of medicine", and emphasizes how essential it is for official and private practitioners to cooperate. It is pointed out, however, that the private practitioner does not apply the principles of preventive medicine to his private practice with the result that the State has been forced to take over these functions. The chapter on "The Practising Physician and the Public Health Department" could be read with profit by all practitioners.

TUBERCULOSIS IN CHILDHOOD.

THE first edition of Dorothy S. Price's "Tuberculosis in Childhood" was published in 1942 and received a favourable review in the columns of this journal. The present edition follows on the same lines as the previous one, but has been revised and brought up to date and several chapters have been rewritten.[1]

An account is given of primary type infection in children and adolescents and emphasis is laid on the value of tuberculin testing and X rays in diagnosis. Various methods of tuberculin testing are described, and the X-ray and other illustrations are good.

The chapter on treatment is rather lacking in detail and may be summed up in the word "rest". No mention is made of the use of streptomycin in the acute lesions, but "B.C.G." vaccination as a preventive measure receives favourable comment. It is not clear that the latter method has been in actual use in Dublin.

This is a useful addition to the literature on tuberculosis in childhood. The book is clearly written and is easy to read. It gives a good account of the Irish school of thought, and is worthy of study by those interested in this subject.

VETERINARY PHYSIOLOGY.

SINCE experimental physiologists work largely with animal material, it would seem easy to write a text-book of veterinary physiology. In fact, the reverse is true, and Professor H. H. Dukes is to be congratulated on the latest edition of the "Physiology of Domestic Animals",[2] for in successive editions he has managed to tell us more and more about animals and less about human beings. Veterinary physiology is essentially comparative and there are still many gaps in our knowledge of the variation to be found both within and between the common domestic species. These gaps are easily ignored by an author who is writing

[1] "Diseases of the Ear, Nose and Throat", by Douglas G. Carruthers, M.B., Ch.M. (Sydney), F.R.A.C.S.; Second Edition; 1948. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall (1941), Limited. 8″ × 5¼″, pp. 356, with illustrations, some of them coloured. Price: 25s.

[2] "Public Health Administration in the United States", by Wilson G. Smillie, A.B., M.D., Dr.P.H., Sc.D. (Hon.); Third Edition; 1947. New York: The Macmillan Company. 9¼″ × 6″, pp. 658. Price: 32s. 6d.

[1] "Tuberculosis in Childhood", by Dorothy Stopford Price, M.D. (Univ. Dublin), with a chapter on Tuberculous Orthopædic Lesions and other Contributions by Henry F. MacAuley, M.Ch., F.R.C.S.I.; Second Edition; 1948. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall (1941), Limited. 7″ × 4″, pp. 228, with illustrations. Price: 25s.

[2] "The Physiology of Domestic Animals", by H. H. Dukes, D.V.M., M.S.; Sixth Edition; 1947. Ithaca and New York: Comstock Publishing Company Incorporated. London: Baillière, Tindall and Cox. 9½″ × 6″, pp. 826, with illustrations. Price: 41s.

on physiology for medical students, but are brought into sharp focus by any attempt to give an account likely to be useful to the veterinarian.

Dukes has maintained a reasonable balance between the various sections of the book. Subjects such as the special senses are not of such importance to veterinary students as to their medical counterparts, and are correspondingly condensed. The sections dealing with metabolism and reproduction, on the other hand, which are essential to the veterinary profession, are large and have been extensively revised. In this respect, the only really disappointing chapter is that dealing with endocrines, which is too short and manages to ignore completely such important recent work as that of Selye and his colleagues on the alarm reaction and the adaptation syndrome in animals. The omission of formulæ of blood pigments and vitamins is also to be regretted; they are given for various hormones, when known, although that of œstradiol on page 741 should have a phenolic ring, with three double bonds instead of the single one shown.

Despite its many good points, the book still leaves a feeling of lack of integration. This lack probably springs from a scientific virtue in its author, whose objective attitude seems to prevent him from making even a pretence at generalization if (as is too frequently the case) the evidence available is barely adequate. Nevertheless, this must sometimes leave the student a little more bewildered than is usually found good for him.

The figures, particularly the numerous graphs, are a feature of the book. They are clearly reproduced and fully explained by legends which make it unnecessary to search the text for a key to their meaning.

ADVANCES IN PÆDIATRICS.

PÆDIATRICS may fairly claim to be the most comprehensive of all the specialties. Volume III of "Advances in Pediatrics",[1] the third annual publication in this series, supports this contention, ranging as it does from the osteochondroses and retrolental fibroplasia to the problems of puberty and adolescence. In all the book contains eight monographs, each written by an authority in his particular field.

In the opening chapter Clement A. Smith discusses the effects of birth processes and obstetric procedures upon the newborn infant, dealing especially with maternal anæsthesia and fœtal anoxia. Drugs, it seems, are used less enthusiastically and more cautiously to initiate the breathing of the apnœic infant than they were ten years ago.

There is a good account of viral hepatitis, in which serum hepatitis (homologous serum jaundice) and infective hepatitis are compared and contrasted.

Psychosomatic problems occur as frequently in pædiatric practice as in general medicine and are discussed by Milton J. E. Senn in a brief section entitled "Emotions and Symptoms in Pediatric Practice".

The best chapter in the book concerns the therapy of epilepsy which, as the writer (W. G. Lennox) remarks, may include anything from placing the patient in a suitable school to a resection of the brain. In order to "fit the treatment to the fit" various types of epileptic attacks are described and the therapeutic and side effects of the most useful drugs.

It is a pity that two long chapters—on abnormalities of sexual development and psychological considerations of puberty and adolescence, occupying 137 pages and concluding with 318 references—have been allowed to spoil the balance of the book. Three or four shorter chapters on different subjects would have been preferable. However, it is pleasing to learn of cryptorchidism that the early enthusiasm for hormonal therapy is now giving way to conservatism because of the large percentage of spontaneous cures which occur during adolescence; and, in another context, that "whereas physical and sexual maturity is attained, failure to attain emotional maturity is one of the basic ills of our present society".

This book is most valuable, not only for its factual information, but because of its considered statements by experts who are in a position to comment upon the available literature in the light of their own experience. The reader can feel that he now has the last word on the particular topics covered.

[1] "Advances in Pediatrics", by various authors; Volume III; 1948. New York: Interscience Publishers, Incorporated. London: Interscience Publishers, Limited. 9" × 6", pp. 376, with illustrations. Price: $7.50.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Shock and Allied Forms of Failure of the Circulation", by H. A. Davis, M.D., C.M., F.A.C.S.; 1949. New York: Grune and Stratton. 9" × 5¾", pp. 612, with 55 illustrations. Price: $12.00.

An account of the nature and treatment of shock for all who are likely to meet it in practice.

"Abdominal Operations", by Rodney Maingot, F.R.C.S. (England); Second Edition; 1948. London: H. K. Lewis and Company, Limited. 9½" × 6¼", pp. 1304, with 468 illustrations. Price: 84s.

Presents a detailed consideration of the technique of modern abdominal operations with the choice of operation and other related matters.

"Failure of the Heart and Circulation", by Terence East, M.A., D.M., F.R.C.P.; Second Edition; 1948. London: Staples Press, Limited. New York: Staples Press Incorporated. 7¼" × 5", pp. 148. Price: 8s. 6d.

Discusses the causes, effects and treatment of heart failure, failure of the coronary circulation and peripheral circulatory failure.

"An Atlas of Bone-Marrow Pathology", by M. C. G. Israëls, M.Sc., M.D., M.R.C.P., with illustrations by D. Davidson; 1948. London: William Heinemann (Medical Books), Limited. Pp. 92, with many illustrations, some coloured. Price: 30s.

Sets out to provide an authoritative, accurately illustrated account of the bone-marrow in health and disease for the guidance of physicians and pathologists.

"Aids to Biochemistry", by E. A. Cooper, D.Sc. (London), F.R.I.C., A.R.C.S. (London), and S. D. Nicholas, B.A. (Oxon.), A.R.I.C.; Fourth Edition; 1948. London: Baillière, Tindall and Cox. 6¼" × 4", pp. 260, with illustrations. Price: 5s.

Intended primarily for purposes of revision for the use of medical and science students.

"Aids to Physical Chemistry", by R. G. Austin, B.Sc. (London), F.R.I.C.; Second Edition; 1948. London: Baillière, Tindall and Cox. 6¼" × 4", pp. 464, with illustrations. Price: 7s. 6d.

For students of medicine, dentistry, veterinary surgery, pharmacy and science.

"Energy and Matter", by R. L. Worrall, M.B., Ch.M., D.P.H.; 1948. London: Staples Press, Limited. New York: Staples Press Incorporated. 8¾" × 5½", pp. 144. Price: 10s. 6d.

Deals with the nature of the physical world.

"The 1948 Year Book of Urology", edited by Oswald S. Lowsley, M.D., F.A.C.S.; 1948. Chicago: The Year Book Publishers, Incorporated. 7" × 4¾", pp. 452, with 79 illustrations. Price: $4.75.

One of the "Practical Medicine Series" of year books.

"The Clinical Examination of the Nervous System", by G. H. Monrad-Krohn, M.D., F.R.C.P.; Ninth Edition; 1948. London: H. K. Lewis and Company, Limited. 7¼" × 4½", pp. 484, with 131 illustrations. Price: 16s.

The book deals with the clinical examination of the nervous system, "nothing more and nothing less".

"Measurements of the Public Health: Essays on Social Medicine", by F. A. E. Crew, M.D., D.Sc., Ph.D., F.R.C.P. (Edinburgh), F.R.S.; 1948. Edinburgh: Oliver and Boyd. 8½" × 5", pp. 268, with illustrations. Price: 18s.

An attempt to find the meaning of statistics provided in the annual report of the Registrar-General for Scotland.

"Cancer of the Esophagus and Gastric Cardia", edited by George T. Pack, B.S., M.D. (New York); 1949. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9¼" × 6¼", pp. 196, with 25 illustrations. Price: 37s. 6d.

A symposium by leading American surgeons reprinted from *Surgery*, June, 1948.

"Adolescence Problems: A Handbook for Physicians, Parents and Teachers", by William S. Sadler, M.D., F.A.P.A. (Chicago); 1948. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 8½" × 5½", pp. 472. Price: 35s. 6d.

Designed to make available the author's experience of over forty years in dealing with adolescents.

The Medical Journal of Australia

SATURDAY, APRIL 2, 1949.

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THE SCOPE OF NURSING.

In the issue of January 8, 1949, an account was given in these columns of a minority report by Dr. John Cohen on the recruitment and training of nurses. Dr. Cohen was the member of a "Working Party" whose report was discussed in this journal in November, 1947. The majority report stated that the problem of staffing the nursing services should be regarded as being partly one of preventive medicine, and it was thought that the problem could be attacked quite as much by reducing the number of patients as by increasing the number of nurses. The committee also reported the result of a "job analysis" of nursing training. Dr. Cohen could not sign the majority report because the committee in his opinion did not take sufficient account either of the relation between the planning of nursing and other health services and the planning of the country's manpower resources as a whole or of the extent to which methods employed in psychological research could provide a scientific basis for determining nursing and medical staff ratios or determining the length of training periods for nurses. At the end of our reference to Dr. Cohen's minority report we stated that underlying all efforts to create an adequate nursing service must be a proper understanding of the scope of nursing in the light of modern developments. The importance of this aspect has been emphasized by a document recently issued by the Russell Sage Foundation in America.[1] The author is Dr. Esther Brown, the Director of the Foundation's Department of Studies in the Professions.

The Russell Sage Foundation was established in 1907 by Mrs. Russell Sage "for the improvement of social and living conditions in the United States of America". Authors of studies carried out for the foundation have the benefit of the criticism and advice of their colleagues, but the responsibility for facts, conclusions and interpretations rests with the research workers alone. Dr. Brown (she is a doctor of philosophy, not of medicine) has written other books for the foundation, and the present volume is described as an attempt to discover how education for an essential profession can be moulded to meet the present needs of society. For all this, the author states in her introduction that though the National Nursing Council invested in her the greatest possible degree of freedom, probably over two thousand persons participated in the formulation of ideas expressed in her report. Some basal decisions were made as a preliminary to the work. First, it was decided to view nursing service and nursing education in terms of what is best for society, not what is best for the profession of nursing as a vested interest. Secondly, it decided that as large an area of the United States as possible should be covered. The third decision was the outcome of discussion on the organization and finance of professional schools of nursing and who should undertake them. With this end in view an attempt was made to determine the probable nature of health services in the second half of the twentieth century and of the nursing services likely to be demanded by them. Inquiry was then made into the kinds of training needed to prepare nurses to render those services. For the benefit of those who will study this subject in detail it may be stated that the chapter headings of the book are as follows: "Extension of Health Services", "Future Demand for Nursing Care", "Differentiation of Nursing Service According to Functions", "Future Role of the Professional Nurse", "Education for Practical and Bedside Nurses", "Education for the Professional Nurse", "Resources for the Future". Throughout, references are made from time to time to conditions existing in parts of the United States. The present intention is to put before readers those aspects which are of possible importance to Australian conditions; figures for the United States will be given only when they are relevant to this presentation.

The first point of importance is that the expanding health services of the future and the growing specialization of hospitals will make an ever-increasing demand on nursing services. One result of this will be that a higher grade of knowledge and efficiency will be required of nurses. In Australia at present nurses are trained in general hospitals and gain a certificate in general nursing at the end of a period of years after they have passed certain examinations. Special training is required for a certificate in midwifery. Again, all hospitals are not provided with pædiatric wards and mental nursing is conducted in special mental hospitals. There is no uniformity in requirements for trainees in the different branches of nursing, and conditions are not uniform in the several States even about what is known as general nursing. In America conditions are different; particularly is this true of the responsibilities that are expected to be shouldered by nurses—for example, anæsthesia is sometimes entrusted to a nurse. Emphasis is laid by Dr. Brown on two grades of nurse—the "professional" nurse and the practical and graduate bedside, or "non-professional", nurse. It is this division, this dual status, around which the interest of Australians in this book will chiefly centre. This "professional" nurse merits some detailed description. An author from the Catholic University of America writes as follows:

> Nursing in its broadest sense may be defined as an art and a science which involves the whole patient—body, mind, and spirit; promotes his spiritual, mental and physical health by teaching and by example; stresses health education and health preservation, as well as ministration to

[1] "Nursing for the Future: A Report Prepared for the National Nursing Council", by Esther Lucile Brown, Ph.D.; 1948. New York: Russell Sage Foundation. 9" x 6", pp. 198. Price: $2.00.

the sick; involves the care of the patient's environment—social and spiritual as well as physical; and gives health service to the family and community as well as to the individual.

Dr. Brown describes the "professional" nurse in the following words:

> These are nurses who will be expected to be fully contributing team members in complex clinical situations, where both technical skill of a high order and psychological and sociological orientation are requisite; in the wide field of community nursing and community health services where prevention of disease and therapy, but more particularly the maintenance of health, are ever-widening goals; in the supervision of the activities of many kinds of assistant and graduate personnel; in planning and administering nursing programs, often within agencies and institutions of great size and complicated organizational structure. Included also are those nurses who must assume responsibility for formulating educational programs; instructing students of nursing, graduate nurses, practical nurses, and other assistant personnel in the content and methodology of nursing, and in what the student and graduate nurse should teach patients individually and in groups and in how this can best be done; carrying on desperately needed research, writing, publication, and consultation related to the improvement and extension of nursing care; planning for the continued growth and development of the nursing profession; and administering those plans in close cooperation with other health services and with the needs of society always in mind.

In view of the high standard set for these "professional" nurses, it is not surprising to see that Dr. Brown recommends that schools of nursing should be created in the future within institutions of higher learning and that they should be made autonomous units vested with the same status as the other professional schools and that schools already in existence, not now fully independent, should be helped to become so. In January, 1946, statistical data were supplied to the National League of Nursing Education by 1214 schools of nursing. Of these, 77, or 6%, reported that they were organized in and controlled by institutions of higher learning—47 were in universities and 30 in colleges. Of the 77 schools, 40 offered a degree programme only, 29 offered both a degree and a diploma programme, and eight offered a diploma only. Having learned what is meant by the term "professional" nurse, we can grasp at once what Dr. Brown means by "practical and graduate bedside nurses", whom she also describes as "non-professional". To what the term "graduate" applies is not quite clear—graduate of what? Of a training school for nurses or of a university? Presumably the university is not intended, though in many circles it would be so understood in this country. There seems to be something grand or superior about the use of the words professional and non-professional. It would be better to use such terms as "administrative" or "academic" or "teaching" for the so-called "professional" nurse and "clinical" for the other, the so-called "non-professional". Dr. Brown quotes Abraham Flexner's criteria of an occupation that has attained a professional status. And it may be claimed that a good case may be made out for the inclusion of "practical and graduate bedside nurses" among those to whom the criteria can be applied. If a university degree in nursing is required of the more highly qualified group, then the term graduate should be applied to that group. In this matter we have to take care that we are not playing with words in a fashion which will get us nowhere, and which may make us more confused than we are.

One statement made by Dr. Brown is of basal importance. She reminds us that hospitals predominantly are operated on the authoritarian principle rather than on that of a cooperative team relationship. "The nursing service is caught between the authority exercised by the medical administration, on the one hand, and the hospital administration, on the other." The nursing service itself also tends to be highly authoritarian. "Hence the individual nurse finds herself with little freedom of movement and of initiative for other than specified duties, even within that service of which she is a part." The nurse is described as primarily a person who takes and carries out orders. There is no doubt that a proficient nurse is always a partner in the team. The physician is wise who allows her, or makes her, to feel that she is. For example, she sees much more of the patient than the doctor does and can act as an unrivalled psychological observer and reporter. Also she has first-rate opportunities for instilling into the patient therapeutic suggestions which the doctor may devise for her use. The type of nurse described as being the promoter of the spiritual, mental, social and physical welfare would need to be a person of keen judgement and would have to be careful not to be chargeable with having usurped the position and functions of the medical attendant. After all, it is the attending doctor who has to accept the responsibility for the conduct of the patient's treatment. The nurse—"professional" or "non-professional"—can further, hinder or even nullify that treatment. If the nurse is to be a co-worker with the doctor in his care of the patient, this will have to be achieved partly by the method of her training and the attitude of the hospital's administrators, partly by the good will and cooperation of the attending physician, and partly by the nurse herself, who will show that she is equal to and fitted for the task.

This short account of the investigation reported by Dr. Brown for the National Nursing Council of the United States, when taken into consideration with the two reports from Great Britain discussed on previous occasions, shows that the nursing problem is real and will become more complex as time passes, for the scope of nursing is expanding. That it calls for examination from the Australian point of view by an Australian body with authority and standing is more obvious than ever. Perhaps some day someone will take notice.

Current Comment.

CORONARY ARTERY DISEASE IN YOUNG MEN.

Disease of the coronary artery system can be judged only by its results on cardiac structure and function; for this reason its diagnosis is difficult, particularly in young people who appear to be well and active. It seems surprising that of 400 men who suffered a coronary "accident" 236 gave no history of previous significant symptoms. These figures emerge from a recent study of men aged from eighteen to thirty-nine years who had served in the 1939-1945 war, and whose medical records are readily available. The research into this material was made by a team representing the clinical, pathological and statistical aspects of the subject, including W. M. Yater, A. H. Traum, W. G. Brown, R. P. Fitzgerald, M. A. Geisler and B. B. Wilcox.[1] Analysis of coronary disease in this selected class has produced some interesting findings. The authors have also reviewed the literature, and have paid special attention to those recorded series in which young

[1] *American Heart Journal*, September, October and December, 1948.

people are mentioned in any detail. They remark that before the war coronary disease was considered rare in men under thirty, and uncommon under forty. Looking back, there seems to be fair justification for this statement; no doubt it is due in part to the comparative delay in the recognition of the importance of chest leads in electrocardiography, and the necessity for a reliable technique. This implies that electrocardiographic research has added appreciably to our knowledge, and this is true. Certainly, as these authors show, the clinical accuracy of the diagnosis, the use of graphic tracings being included in the word "clinical", is decidedly not high. In fact they find that only 58% accuracy was attained by comparison with the post-mortem findings. The expression "coronary artery disease" in this inquiry included only advanced manifestations of arterial disease in the coronary system, evidences of well-established myocardial degenerative changes associated therewith, and also cardiac infarction. In those patients who recovered the diagnosis required confirmation by undoubted evidence of the known effects on the heart of coronary inadequacy of a progressive type, culminating in a clinical crisis of some kind. From the literature this group of observers have drawn 744 examples, of which 597 were in males: from their own series they have produced 868 cases with 450 autopsies. These large numbers are surely due to more adequate diagnostic sifting than to a rising frequency.

Not much light was shed on ætiology; it appears that men with a familial history are more likely to have coronary disease, that it seems to affect men from middle class families rather than the poorer classes, that moderate smoking had no effect, but that smoking more than ten cigarettes a day appeared to increase the risk, and that alcohol had no obvious influence one way or another. The age incidence within the group increased rather sharply at twenty-eight and still more sharply at thirty-three, the peak being at thirty-eight years. Occupation within the series was not significant. The height-weight ratio was of interest, because, though at first sight there seemed to be a loading of the higher weight groups, comparison with control series showed that there was no significant difference. Little of importance appeared from the study of blood pressures. As might be expected, the frequency of coronary disease increased greatly when the diastolic pressure rose to 90 millimetres of mercury, when contrasted with a figure ranging from 70 to 89. This merely reflects the significance of hypertension. Those who like to speculate may reflect on the possible reasons why the blood pressure of those who survived was greater than in those who died. The controversial subject of bodily activity and its bearing on coronary emergencies was also examined. Statistical analysis of the case histories showed that the number of cardiac attacks which occurred during a period of strenuous activity was more than twice that which might have been expected in proportion to the time occupied in such activities. Much the same figures were found when the degree of physical activity was moderate or mild. During sleep only one-third of the expected attacks took place. This last finding upsets an impression which has gained some ground that the hours of rest were in a certain inaccurate sense a time of hazard. The symptomatology is fully covered in this report, but it needs no special mention here, as it conforms to the universally accepted clinical picture. The suddenness of the onset of pain is stressed, also the frequency of the signs of shock, dyspnœa and nausea or vomiting. To the list given perhaps it might be worth adding the recognition by the patient that the pain is different from other varieties he has previously felt, even though he may call it by a familiar name. This point may not sustain statistical analysis, yet it is sometimes striking in clinical experience.

Not much can be said here about the electrocardiographic findings; these need skilled interpretation, and despite the popularity of the use of the electrocardiograph, it is not unfair to point out that full knowledge of the inner meaning of these tracings has lagged behind the application of the method in clinical medicine. Other points raised in this article are the frequency with which the physical examination of the heart during an emergency reveals no abnormality; the figure found in this series was 60%. The blood pressure likewise does not always fall. Among the survivors the commonest residual signs were fatigue and dyspnœa. It is not out of place to point out that these are also common symptoms in conditions of coronary insufficiency, though no occlusion may have occurred. The pathological findings showed that of 374 hearts weighed 52 were enlarged; 114 showed gross infarcts, but 336 had had no gross infarctions. In 232 hearts almost complete occlusion was found of part of one or more of the coronary arteries. This is worth some reflection when a patient is assured that his heart is sound, though it is not suggested that any other course could be taken when a full examination discloses no evidence of disease.

Many other interesting matters are discussed and analysed in this study. Perhaps the fact that the patients were all relatively young points the inquiry; their age must be taken into account when the place of activity in the causation of occlusive attacks is being considered. It will be agreed that the growth of knowledge in this subject has been remarkable, and surely it will continue to enlarge our clinical horizons.

ELECTRONIC MAPPING OF THE ACTIVITY OF THE HEART AND BRAIN.

IT was only to be expected that sooner or later some of the devices employed in radar would be applied to the investigation of the interior of the human body. This has definitely come in the electronic mapping of heart and brain reported by S. Goldman, W. E. Vivian, C. K. Chien and H. N. Bowes.[1] The method used is not, however, based on any sort of echo, but is really a new way of demonstrating the changes in electric potential arising from cardiac and cerebral activity and so may be designated as an extension of electrocardiography and encephalography. The principle can be best explained by describing the first application of the technique to the heart. The fixed electrodes attached to the anterior thoracic wall are placed in four vertical rows of four each, sixteen in all, in the second, third, fourth and fifth intercostal (or interchondral) spaces. The first vertical row is in the left mid-clavicular line, the second is half-way between this and the left margin of the sternum where the third vertical row is placed, the fourth is on the right sternal margin. These sixteen electrodes are connected with sixteen pick-up grids arranged close together and placed in front of a single anode. An electronic beam is then swept rhythmically or "scanned" across the grids. The potential on the grid determines the amount of beam sent to the anode. A standard type of cathode ray tube with a phosphorescent screen is "scanned" in synchronism with the grid system and the signal current from the anode after suitable amplification is applied to the intensity grid of the cathode ray tube. Each horizontal line of grids receives four scanning signals before the beam passes to the next line, and so all sixteen are eventually involved in a regular sequence. In this way there is presented a two-dimensional display of the distribution of electric potential over the selected surface of the chest. The brightness at a given position on the screen is proportional to the potential of the corresponding point on the chest. The need for refinements in technique was soon apparent on account of certain drawbacks, particularly the absence of perfect equality of sensitiveness in the sixteen grids; further the changes in potential from the skull leads were rather too quick for the original rate of scanning. These and other difficulties were eventually surmounted by the substitution of an electronic circuit commutator for the grid system, but to understand the complicated apparatus demands more than a bowing acquaintance with ultra-modern physics. Whether this new technique can render a real service to the study of heart and brain the future will disclose. What has been published is insufficient to allow anything like a proper assessment of its value to be made; indeed the main impression given is that the inventors are more concerned with the method than with the results, a very common attitude in American institutes not directly associated with physiology and pathology but conducting research on medical subjects.

[1] *Science*, December 24, 1948.

Abstracts from Medical Literature.

DERMATOLOGY.

Treatment of Localized Scleroderma.

J. M. Flood and J. H. Stokes (*Archives of Dermatology and Syphilology*, May, 1948) report seven cases of localized scleroderma. The results of treatment with bismuth compound were excellent in two cases, good in four and fair in one. All of the patients showed improvement after receiving a bismuth compound. "Muthanol" and "Iodobismitol" were used, but according to other reports bismuth subsalicylate is also effective. The mode of action of the bismuth compound is not known, but as in its action in other diseases, such as *lupus erythematosus* and *lichen planus*, there is some unknown non-specific effect. It is considered advisable to give a course of treatment lasting ten to twelve weeks, repeating it two or three times if necessary. Apparently some patients will require a total of 50 to 75 injections of bismuth compound before satisfactory results can be obtained.

Topical Applications of "Benadryl".

T. H. McCavack, P. Schulman, R. Schutzer and H. Elias (*Archives of Dermatology and Syphilology*, March, 1948) state that the powerful antihistamine action of "Benadryl Hydrochloride" has been repeatedly demonstrated in animals and in human beings with and without allergic diseases. The frequency of side effects of the drug, when given in adequate amounts by mouth, led the authors to investigate its topical use for diseases in which cutaneous manifestations of a probably allergic nature were present. In 63 cases the topical application of 2% and 5% concentration of "Benadryl Hydrochloride" in ointment bases reduced or completely destroyed the response of the skin to intradermally applied histamine. "Carbowax 1500" monostearate, an oil-in-water emulsion, appeared to be the best of the bases employed. Seventy-four patients with itching dermatoses were treated with ointments containing "Benadryl"; 44 obtained complete relief from itching and 18 were improved. Six patients experienced some discomfort from the application of the ointment, particularly when a 5% concentration was used. However, in four cases this lasted not more than five minutes. In the other two, it was due to the base employed in one and to an aggravation of a weeping eczematous disease in the other. No systemic side effects were observed.

Mosaic Type of Plantar Wart.

A. H. Montgomery and R. M. Montgomery (*Archives of Dermatology and Syphilology*, March, 1948) state that the mosaic wart has clinical characteristics which distinguish it from the single (traumatic) and mother-daughter (epidemic) types of plantar warts. The warty patch is horny and granular with an irregular border and varies in width from one millimetre to ten centimetres. Comparatively painless, the patch is located under the pressure points and may extend onto the toes and behind the heel. It is often mistaken for callus and may be present for years. Paring reveals the multicellular nature of the patch. Each consists of many small individual cores so closely grouped that they resemble a mosaic pattern. Cure of the average patient required eleven and two-tenths treatments in the authors' series. Four chemical preparations are used: (*a*) silver nitrate solution one gramme to one millilitre of distilled water, (*b*) a saturated solution of monochloracetic acid, (*c*) 40% salicylic acid plaster, and (*d*) 60% pulverized salicylic acid ointment. The area is pared until the capillary tips are visible or bleeding. Capillary bleeding is stopped with the silver nitrate solution. The wart is swabbed lightly with saturated solution of monochloracetic acid or silver nitrate solution. Salicylic acid plaster, shaped to the size of the wart, is applied. The same treatment is given to satellites. For more horny lesions the plaster is fortified with a thin coating of 60% salicylic acid ointment. For a deeper reaction saturated solution of monochloracetic acid, instead of silver nitrate, is rubbed in. The plaster is held in place with adhesive plaster. The area is kept dry during treatment. The lesion is pared down weekly to active warty tissue, and the aforementioned steps are repeated. When the area thins, silver nitrate solution alone is used. Every vestige of wart must be removed to avoid recurrence. For severe reactions following the application of monochloracetic acid the medication is removed, a wet dressing is applied and tissue is pared down to release the subwart fluid. Foam rubber pads, placed posterior to the warts, are used to relieve pressure. Observation is continued for a month or two after apparent cure has been obtained.

Hereditary Predisposition in Vascular Disturbances of the Leg.

Alice Carleton (*Archives of Dermatology and Syphilology*, April, 1948) discusses the literature bearing on the occurrence of an hereditary factor predisposing to local vascular disturbance and ulceration and points out a striking family history in two cases of varicose ulcers occurring in sisters. Two cases of pseudoscleroderma of the legs also are described occurring in a mother and daughter. The author states that there is still much doubt as to the exact interrelationship of scleroderma, scleropoikiloderma, *acrodermatitis atrophicans*, macular diffuse *dermatitis atrophicans* and numerous unclassified *formes frustes* that are encountered. They think that it is premature to do more than speculate on the presence of a local predisposition of the tissues to atrophic changes or a local vascular instability acting alone or affecting the nerves of the skin; but that the hereditary factor in these diseases deserves further inquiry.

Cutaneous Hypersensitivity to Penicillin.

B. Phillips (*The British Journal of Dermatology and Syphilis*, November, 1948) reports a case of cutaneous hypersensitivity to penicillin. The patient had been sensitized by applications of penicillin cream to a varicose dermatitis when the cream was used for a fortnight. Three months later an acute generalized eczematous allergic reaction was precipitated by a single application of penicillin cream. Two months after full recovery patch tests carried out with various penicillin preparations produced an intense vesicular erythematous reaction in all cases. The author states that manifestations of cutaneous hypersensitivity to penicillin and to sulphonamides bear a striking resemblance. An important difference is that allergic dermatitis cannot be precipitated by initial application of sulphonamides as it can be with penicillin. Exposure to common antigenic fractions in other related fungi can perhaps sensitize a person to the whole group, including penicillin, so that the first exposure to penicillin may precipitate the reaction. This may explain reactions to penicillin employed locally in the treatment of various skin diseases. The authors recommend that patients for whom it is proposed to use penicillin should be questioned as to previous reactions to fungi, and if necessary should be tested intracutaneously with 500 to 1000 units of penicillin; for patients with a history of chronic fungous infection special local care should be given to areas subject to mycotic infection. The eczematous reaction to penicillin is less damaging to the skin than that produced by sulphonamides, and recovery is more rapid.

Brilliant Green and Chronic Ulcers of the Skin.

A. W. Feinberg (*The New England Journal of Medicine*, October 21, 1948) reports a limited series of cases of chronic ulcers of the skin treated with a 2% solution of brilliant green in distilled water. After thorough cleansing of the ulcer with hydrogen peroxide and alcohol, the dye was painted on daily until healing was well progressed —generally about seven to ten days. It was then applied every other day until healing was complete. The ulcer was covered with a dry sterile dressing after each treatment. The patient was advised to rest as much as possible and to sleep with the affected extremity elevated. The author states that brilliant green is bactericidal for many of the common pathogenic bacteria, has a high stimulating effect on the growth of epithelium and granulation tissues, and counteracts the malodorous discharge common in this type of case. It is completely non-toxic, does not irritate tissues and is low in cost.

UROLOGY.

Carcinoma of the Prostate.

J. B. Wear and A. P. Schoenenberger (*The Journal of Urology*, April, 1948) present a review of 400 patients with prostatic carcinoma, treated over a period of twelve years. They have been able to achieve a follow-up of 383 patients out of the 400. Comparison of the various methods of treatment is difficult since the indications for various types of therapy differed so much. Relative survival figures are also misleading, since, for instance, among the group of 103 patients receiving only endocrine therapy metastases were already present in 50%, while among the endoscopic resection group of 145, metastases on admission were present in only 21%. In a small proportion of patients prostatic carcinoma is a very slowly advancing disease, and the method of treatment which happens to be used is given unfair credit because

of the long survival. On the other hand, the disease is associated with an advanced age group subject to degenerative diseases; but once the diagnosis of prostatic carcinoma is made, that is recorded on the death certificate as the cause of death, even though it may not be the true cause. During the first part of the twelve-year period, the modern methods of antiandrogen therapy and endoscopic resection were not used, and it was noted that suprapubic cystostomy did not prolong the life of patients. However, the patients had to contend with the apparatus, and were neither comfortable nor happy. Irradiation treatment was given, but this has now been relegated to a minor role, the major therapeutic methods being orchidectomy and the administration of œstrogens. Endoscopic resection prolonged the average life of patients. There was a 9% survival for five years in the resection group. A few patients survived up to nine years, and all in this survival group of five to nine years were symptom free. Radical perineal prostatectomy is unfortunately applicable only to about 4% of patients with prostatic carcinoma.

Renal and Ureteric Calculi.

H. K. SANGREE (*The Journal of Urology*, May, 1948) has followed up for periods varying from one to ten years fifty consecutive subjects of renal and ureteric calculi. Stone recurred after operation in 14% of the patients. Nephrectomy was necessary in 18% of cases on account of the large size of the stone, with consequent infection and renal damage. An endocrine cause of the calculosis (hyperparathyreoidism) was proved in only one case of the fifty. Dietetic deficiencies or avitaminosis have played only a minor part in the series.

Non-Hormonal Adrenal Cortical Carcinoma.

Z. R. COTTLER (*The Journal of Urology*, September, 1948) states that a survey of recent literature on tumours of the adrenal cortex reveals a decided emphasis on hormone-producing neoplasms. This is readily understood, since these tumours evoke striking changes externally, and equally dramatic changes follow their extirpation. On the other hand, reports on non-hormonal tumours are scarce, since they seem to be comparatively rare, there is an absence of early symptomatology and, in the late stages of the disease with metastases, confusion may arise as to the origin of the primary growth, for the adrenal is often the site of secondary growths. The non-hormonal type of adrenal carcinoma is usually discovered in its late stages, when made manifest by a large mass, vague pain, malaise and anorexia, and, like a "silent" hypernephroma, by signs and symptoms due to a metastasis in a lung or other organ. These neoplasms are seen practically only in adults aged over thirty years, about equally in either sex. So far, the treatment has been surgical extirpation, when possible, followed by high-voltage irradiation to the operative site, and to the metastases as they arise. Since these tumours often metastasize before producing symptoms, the prognosis is usually poor. The author describes a case discovered accidentally in a woman, aged fifty years, in hospital for a prolapse operation. The remarkable feature is that the patient is still alive and comfortable five years after extirpation of the neoplasm, and even though metastases were discovered two and a half years after the operation. The growth was an adenocarcinoma and was attached to the kidney. As the patient's condition became grave during the operation, time was saved by removing the kidney with the growth. The operation was performed on account of the result of excretion urography, which revealed considerable pushing down of the right kidney by a large mass above it. The kidney was also twisted by the pressure. The author considers that, had he used perirenal air insufflation in diagnosis, he would probably have made a correct pre-operative diagnosis of adrenal neoplasm. Another interesting feature is that severe pre-operative hypertension has been reduced to normal blood pressure levels ever since the operation. The author thinks that the twisting of the kidney caused relative renal ischæmia with consequent hypertension —the Goldblatt phenomenon—so that it is fortunate, perhaps that the kidney was removed. An adrenal hormonal ætiology for the hypertension can be eliminated, since extensive metastases are present, and the blood pressure remained low.

Vesico-Sigmoidostomy in Vesical Exstrophy.

R. RIZZI (*The Urologic and Cutaneous Review*, September, 1948) states that modern attempts at operative relief of vesical exstrophy are concerned with some form of deviation of the urine to the intestine. He has used the second method of Coffey with some success. He finds, however, that in young subjects this is a severe ordeal, and has evolved a modification of Maydl's and Moynihan's method, so that the bladder can be safely and successfully anastomosed to the sigmoid colon. He does not disturb the region of the trigone, and is at pains to interfere as little as possible with the vascular and nervous supply of the bladder. Catheters are placed in the ureters and kept there till just before final closure. A mid-line laparotomy incision is made 2·5 centimetres above the upper vesical edge. Two fingers are placed in the peritoneal cavity to protect intestinal coils and aid bladder mobilization. The incision is now continued around each vesical edge for about three-quarters of the way on each side, peritoneum on the back of the bladder being cut through as well. Below this point on each side the incision goes through only the mucosa and part of the muscle of the bladder. This partial incision allows enough mobilization for suture of the lower part to the sigmoid colon, and it allows preservation of the main nerves and vessels entering the bladder. In this and the subsequent manœuvres, one takes care to keep away from the ureters, which are delineated by catheters as stated above. Now a loop of sigmoid is brought up to the left side of the bladder, and held there in the grasp of a pair of long intestinal forceps. A serosa-to-serosa suture unites the side of the bladder with an appropriate line on the colon wall. One now incises the colon longitudinally for the same length as the suture just placed. The intestinal lumen is cleaned, and the contiguous (deeper) seroso-mucoso-muscular layers of bladder and gut are sutured together. In the lower part of this suture the ureter is particularly avoided. A small injury to the ureter might cause a urinary fistula, or even peritonitis. The suture through all layers is now completed on the superficial aspect, the ureteric catheters being withdrawn just before closure of the gut and bladder is effected. A final seroso-serous invaginating suture is placed, and the intestinal forceps are then removed. The superficial peritoneal layer is difficult to suture at the lower end of the wound, but it may be detached and mobilized at the top and sides of the wound; at the lower end of the wound the peritoneum will have to be sutured to the muscle wall of the lower part of the bladder. Likewise, suture of the separated muscles and skin is difficult or impossible in the lower portion. A small concave area remains uncovered, and this is lightly filled with gauze. A rectal tube is left indwelling for eight days to avoid colonic distension. No laxative is given until after eight days. Streptomycin and sulphaguanidine are given before and after operation. The author has had four excellent results in four cases in children aged respectively four, seven, eight and twelve years. The great value of this type of operation, which respects the main nerve and blood supply to the bladder and leaves the intramural portion of the ureters intact, is that the protective function of the latter portions is maintained even though the ureters now open into the bowel.

Wilms's Tumour.

W. J. DAW (*The Journal of Urology*, July, 1948) reports seven cases of Wilms's tumour, or renal embryoma, encountered between 1937 and 1945. Another name for this highly malignant disease of early childhood is embryonal adeno-myo-sarcoma of the kidney, and this description suggests the cellular formation. The most characteristic cell is a mesodermal fibroblast, which may differentiate into renal tubules and glandular structures. In some of these growths striated muscle fibres are found. X-ray treatment causes a loss of mesodermal elements, and a replacement with better differentiated structures. All seven of the author's patients were aged under four years. There is no predominance of sex or side. The clinical characteristics are a very large mass on one side, rounded, and not movable or tender. Pallor, pyrexia, peevishness and hæmaturia are present. One must be careful to differentiate a Wilms's tumour from a large hydronephrosis. The treatment is nephrectomy as soon as the diagnosis is confirmed. Most urologists are unconvinced that X-ray therapy alone will cure Wilms's tumour, since only occasional five-year cures are recorded. The method usually accepted is to shrink the tumour with pre-operative X-ray therapy, perform nephrectomy and follow with post-operative irradiation. The preliminary irradiation should not occupy more than two to three weeks. The trans-peritoneal approach is the best, and the chances of survival can be fairly well estimated by the appearance of the kidney pelvis, the renal vein, the surrounding glands and the perirenal tissue. Two of the author's patients presented advanced metastases and died without operation. Of the five patients operated on, two died of metastases, and three children have survived for six, five and two years respectively.

British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch of the British Medical Association was held on December 2, 1948, at the Saint George Hospital, Kogarah. The meeting took the form of a series of clinical demonstrations by the members of the honorary medical staff of the hospital.

Pulmonary Tuberculosis Treated by Streptomycin.

Dr. C. G. Bayliss presented a male patient, aged fifty-two years, who had been admitted to hospital on February 2, 1948, suffering from severe hæmoptysis. He had had a cough for years, and some months previously X-ray examination of his chest had indicated the presence of a small chronic tuberculous lesion in the apex of the right lung. On his admission to hospital he was very distressed, sweating and dyspnœic, with a rapid pulse rate. The hæmoptysis continued for about six days. Tubercle bacilli were found in the sputum. X-ray examination of the chest indicated the presence of an old tuberculous lesion of limited extent in the apex of the right lung, where a small cavity could be seen. Throughout the right lung there was also extensive mottling, some of which might have been caused by retained blood; but a large amount of the mottling was considered to indicate an acute spread of the disease. The condition was diagnosed as hæmoptysis accompanied by an acute spread of the disease occurring in a patient who previously was suffering from a chronic tuberculous lesion of limited extent. The administration of streptomycin in a dosage of 0·5 gramme given intramuscularly twice a day was commenced on March 6, 1948. This dosage was continued for eight weeks and then reduced to 0·5 gramme once a day for a further four weeks. X-ray examination on March 25 indicated no essential change in the condition of the right lung. However, in a specimen of sputum collected on March 31 no tubercle bacilli were detected, either by direct smear examination or by culture. The appearances in an X-ray film taken on April 30 suggested that some clearing of the acute spread had occurred, and the appearances in a film taken on June 9 showed the presence of the residual chronic lesion in the apex of the right lung. The signs of the acute spread had completely disappeared. Dr. Bayliss commented that in the case under discussion streptomycin seemed to have been the significant agent in causing resolution of an acute spread of tuberculous disease following hæmoptysis. The drug had had no effect on the basic chronic lesion.

Pulmonary Tuberculosis Treated by Phrenic Crush and Pneumoperitoneum.

Dr. Bayliss's second patient was a woman, aged twenty-six years, who in April, 1945, had been diagnosed as suffering from pulmonary tuberculosis affecting the upper zone of the right lung, in which a cavity was obvious. She was treated in the Thirlmere Sanatorium from May, 1945, until December, 1946. In June, 1946, artificial pneumothorax was induced, but inoperable adhesions prevented effective collapse of the lung, and the treatment was abandoned in February, 1947. At that time a large cavity was present in the mid-zone of the right lung, with a moderate amount of infiltration in the lower zone and a moderate amount of fibrosis extending upwards from the hilum. During the next twelve months the patient was kept at rest, and a definite improvement occurred; but in February, 1948, a residual cavity was still present, and the sputum contained numerous tubercle bacilli. Phrenic crush was carried out on April 2, 1948, and the induction of pneumoperitoneum was commenced on April 3. At the time of the meeting the patient was clinically very well. The right dome of the diaphragm had been elevated about four inches, no evidence of cavitation could be seen and the lesion appeared to be under control. It was intended to continue treatment for at least a further two years.

Frontal Lobe Abscess.

Dr. E. R. Figtree discussed the case of a male patient, aged seventeen years, who had been admitted to hospital complaining of severe headache and pain over the left eye of one week's duration, and of confusion of four days' duration. He had had disease of the spine four years previously, which was believed to have been early Pott's disease. On examination of the patient, he was confused and suffering from photophobia. Bilateral ptosis was present, more pronounced on the left side. The pupils were unequal, the right being larger than the left, and reacted to light. The *fundi oculorum* appeared to be normal. Neck rigidity and Kernig's sign were present in pronounced degree. All reflexes were normal. The throat showed signs of slight infection, and the right ear drum was slightly injected. Apart from tachycardia, no abnormality was found in the respiratory and cardio-vascular systems. Lumbar puncture produced clear cerebro-spinal fluid under a pressure of over 300 millimetres of fluid; 188 leucocytes, mostly polymorphonuclear, were present in each cubic millimetre; the protein and chloride contents were 50 milligrammes and 700 milligrammes *per centum* respectively. No organisms were found, either from direct examination or from culture. Penicillin (15,000 units) was given intrathecally. The left frontal sinus was opened and the frontal lobe exposed. Acute empyema of the sinus and a left frontal abscess were revealed. A drainage tube was left in the abscess. On the following day the patient was semicomatose; a little blood-stained material was draining from the wound. Pus was withdrawn from the tube, and was found to contain pnemococci and brain tissue. The left pupil was dilated and fixed. On the next day the patient could not be roused. Periodic breathing and muscular twitching were observed, both plantar reflexes were extensor in type, left knee clonus was present, and the left knee jerk was greatly increased. Subsequently twitching of the right side of the face, the right arm and the right leg occurred, leading to an epileptiform convulsion, the right pupil being dilated and fixed. The temperature rose to 102° F. Four days after operation the patient's condition was much improved, his temperature had fallen to 99° F., he could be roused, and the twitching had ceased. The pupils reacted sluggishly to light. Very little rigidity remained. The cerebro-spinal fluid was clear, and under a pressure of 150 millimetres of fluid; three leucocytes per millilitre were present. The hæmoglobin value was 62%. On the following day there were further signs of improvement. The ankle and knee jerks were absent, the right plantar response was flexor, and the left plantar response was extensor. Both pupils reacted to light. A day later, however, signs of hypostatic pneumonia developed, the patient became comatose and the temperature fell below normal. The cerebro-spinal fluid was still clear and under a pressure of 65 millimetres of fluid. The patient's condition from then on rapidly deteriorated, and death resulted.

Tuberculous Meningitis.

Dr. Figtree's second case was that of a male patient, aged nineteen years, who had been admitted to hospital on October 17, 1948, complaining of frontal headaches of six months' duration, more severe during the preceding two weeks, giddiness, vomiting and "trouble" with the left eye of one week's duration and mental confusion present for one day. On examination of the patient he was confused, restless, incoherent and unable to cooperate. The pupils were normal, and there was no evidence of papilœdema. The right eye was turned in, and the left side of the face was twitching. All reflexes were normal, except that the abdominal reflexes were absent. There was no evidence of muscular paresis or alteration of tone. Kernig's sign and neck rigidity were both present. Lumbar puncture produced opalescent cerebro-spinal fluid, slightly blood-stained as the result of trauma, and under greatly increased pressure. The Queckenstedt sign was present. The fluid contained a moderate number of leucocytes (mainly lymphocytes), and 200 milligrammes of protein and 720 milligrammes of chlorides *per centum* respectively. No organisms were found from direct examination or from culture. For the next two days the patient remained comatose. An intrathecal injection of penicillin was given. Albumin and acetone appeared in the urine. The patient appeared to be suffering from severe frontal headache, and a left frontal abscess was diagnosed. Exploratory operation, however, revealed no sign of abscess, and X-ray examination of the frontal sinuses revealed no abnormality. On October 20 the state of the cerebro-spinal fluid was unaltered, but the patient was completely conscious and fairly cooperative. The right pupil was larger than the left, and both reacted sluggishly to light. Bilateral sixth nerve palsy was present, and neck rigidity was still pronounced. A temporary further improvement occurred, but by October 22 his condition was again worse; palatal palsy had developed and all reflexes were absent on the left side. The state of the cerebro-spinal fluid was suggestive of tuberculous meningitis, although no tubercle bacilli were found. Streptomycin therapy was commenced. By October 25 the patient's condition was much improved. The cerebro-spinal fluid pressure was 300 millimetres of fluid, and the Queckenstedt sign was absent. X-ray examination of the chest strongly suggested pulmonary tuberculosis affecting the middle and lower zones of the

right lung. On October 26 the cerebro-spinal fluid pressure had fallen to 90 millimetres of fluid and the patient's condition was still improving. However, on October 27 he developed hypostatic pneumonia, his condition rapidly deteriorated and he died.

Acholuric Jaundice.

Dr. J. C. English presented a male patient suffering from acholuric jaundice complicated by ulcerative colitis, who had been admitted to hospital in November, 1947, with the provisional diagnosis of carcinoma of the stomach. He had a history of recurring diarrhœa for four months with the loss of one stone in weight. X-ray examination with an opaque meal had revealed no abnormality in the stomach and duodenum. Examination of the fæces had revealed no signs of parasites or pathogenic organisms. Sternal biopsy revealed the presence of an erythroblastic reaction of normoblastic type. Blood counts indicated the presence of normocytic or macrocytic anæmia with leucocytosis; the proportion of reticulocytes was increased to 14%. The serum bilirubin content was slightly increased. X-ray examination revealed no abnormalities of the long bones or of the chest. No evidence of new growth was found at laparotomy. Treatment with refined liver extract, vitamins, iron and a diet of high protein content had no beneficial effect on the patient's anæmia. When his diarrhœa had ceased, he was discharged from hospital and instructed to take folic acid, because the blood count had revealed an increased mean corpuscular volume. The final diagnosis at that time was macrocytic anæmia. Five weeks later the patient was readmitted to hospital with diarrhœa and debility. In the interim splenic enlargement had been noted at the out-patient department, and the patient had become icteric and pale in spite of the folic acid. Sigmoidoscopy revealed the presence of ulcerative colitis. The presence of severe normocytic anæmia and leucocytosis was apparent from a blood count, the result of sternal biopsy being as before. No significant information was obtained from bacteriological and parasitological examination of the fæces or from Wassermann and Kahn tests. The serum bilirubin content was increased to 2·4 milligrammes *per centum*, and the serum alkaline phosphatase content was also increased. The results of liver function tests were normal. Occult blood was present in the fæces. The fragility of the erythrocytes was slightly increased. The patient was treated with repeated blood transfusions, a diet of high protein content including amino acids, hog's stomach, iron and vitamins, as well as the parenteral administration of penicillin and local bowel lavage. It was thought that the hæmolysis might have been due to hæmolytic streptococcal infection of the bowel ulcers. Transfusion appeared to be the only treatment of value. The hæmoglobin value began to rise after three months, and was normal on the patient's discharge from hospital. The diagnosis at that stage was ulcerative colitis with hæmolytic anæmia.

The patient was readmitted to hospital two months later when laboratory evidence was obtained of acholuric jaundice: anæmia, spherocytosis, reticulocytosis, increased erythrocyte fragility, raised serum bilirubin content and increased fæcal and urinary urobilinogen content. As on the patient's previous admission to hospital, sigmoidoscopy revealed ulceration of the bowel. Splenectomy was performed in September, 1948, and the patient was discharged from hospital clinically well six weeks later, with a hæmoglobin value of 11·2 grammes *per centum*, and with his bowel healed; the serum bilirubin level was still slightly raised. The diagnosis was ulcerative colitis and acholuric jaundice. Almost immediately after his discharge from hospital the diarrhœa recurred, but was not severe. Approximately three weeks later sigmoidoscopy revealed no abnormality, the hæmoglobin value of the blood was 13·8 grammes *per centum*, some spherocytes being present, and the serum bilirubin level was still slightly raised.

Subacute Bacterial Endocarditis.

Dr. English then showed two patients who had been successfully treated for subacute bacterial endocarditis. The first patient, a male, aged seventeen years, with a history of five previous attacks of rheumatic fever and diphtheria and scarlet fever in childhood, was admitted to hospital with a provisional diagnosis of rheumatic carditis, the possibility of subacute bacterial endocarditis being kept in mind. His heart was grossly enlarged, with signs suggesting mitral regurgitation and aortic incompetence. The spleen was thought to be slightly enlarged. There were no embolic phenomena. He was treated with ordinary doses of penicillin and salicylates, and his temperature quickly became normal; but he soon suffered relapse. A culture of *Streptococcus viridans* was grown from his blood. A course of penicillin (100,000 units every three hours) proved effective, and in less than two months he was free of symptoms and was discharged from hospital to Primrose House Convalescent Hospital.

The second patient was a married woman, aged thirty-eight years, with a past history of three attacks of rheumatic fever. On her admission to hospital, a diagnosis was made of mitral stenosis and fibrillation and unresolved lobar pneumonia. The possibility of subacute bacterial endocarditis was thought of, but no confirmatory signs were found. The patient was treated with normal doses of penicillin and digitalis. Her elevated temperature subsided immediately, but it again rose approximately three weeks later, and once more responded to penicillin. She appeared to progress slowly, with slight relapses during the next two months. Then she complained of painful lumps on her fingers, and was found to have Osler's nodes, an enlarged spleen and petechiæ under the nails. No organisms were grown from her blood, but a diagnosis of subacute bacterial endocarditis was made, and a course of 100,000 units of penicillin every three hours for twenty-eight days was begun. Her temperature settled within ten days, the spleen became smaller and there were no signs of any further embolic phenomena. She was discharged from hospital, apparently well, but with impaired exercise tolerance.

Lumbar Sympathectomy.

Dr. A. C. Thomas presented four patients to illustrate the use of lumbar sympathectomy in the treatment of peripheral vascular disease. The first patient, a male, aged fifty years, was suffering from *endarteritis obliterans*. After lumbar sympathectomy trophic ulcers developed on the right foot, and subsequently gangrene under the right great toe and over the corresponding metatarso-phalangeal joint. This was regarded as being arteriosclerotic in origin. The patient left hospital against medical advice when the toe was sloughing.

The second patient, a male, aged eighty-three years, had been suffering from senile arteriosclerotic gangrene of the left foot with coldness of the foot and intermittent claudication of the left leg. After left lumbar sympathetic block, the patient was much relieved and the leg became warm. Lumbar sympathectomy was then carried out, and at the time of the meeting the foot was warm and comfortable.

A male patient, aged thirty-five years, was suffering from *thromboangiitis obliterans* involving particularly the left leg, which was œdematous, with an area of moist gangrene on the great toe and a small area on the side of the foot. The left foot was cold and the pulsation of the *dorsalis pedis* artery was impalpable. After removal of the third and fourth lumbar ganglia the leg felt much warmer. When the patient was discharged from hospital eleven days after operation, his condition was regarded as satisfactory; the left foot was warm and the pulsation of the *dorsalis pedis* artery was palpable. The left great toe was still gangrenous, with a line of demarcation at its base.

A male patient, aged fifty-six years, had suffered from intermittent claudication in the left leg for twelve months, and from a black ulcerating lesion on the left fourth toe for one month. The leg was pale and painful when elevated, reddish-blue and less painful when dependent. No improvement followed left paravertebral block with "Novocain". Periarterial sympathectomy was then carried out for a distance of half an inch in the middle of the thigh. The vessel was found to be calcified and without pulsation. The operation produced no apparent improvement, and after excision of the gangrenous left fourth toe the patient was discharged from hospital. He was readmitted approximately one month later, the leg being in much the same condition. The second and third lumbar sympathetic ganglia were excised, with much improvement in the condition.

Actinomycosis of the Colon.

Dr. Thomas then presented a female patient, aged forty-three years, who, between two and three months after an operation for acute gangrenous appendicitis, was readmitted to hospital with a tender mass in the right side of the abdomen at the level of the umbilicus. At laparotomy a large fungating mass was found at the hepatic flexure of the colon, adherent to the lateral abdominal wall. A provisional diagnosis of carcinoma was made, and she was discharged from hospital. About a fortnight later she was readmitted with a discharging abscess over the mass in the right side. Examination of the pus revealed the presence of actinomycosis. She was treated with 400,000 units of penicillin twice a day, and discharged from hospital one month after her admission.

Carotid Body Tumour.

Dr. Thomas's next patient, a male, had had a painless swelling on the left side of his neck, increasing in size, for five years. It was two inches in diameter, situated behind the middle third of the sterno-mastoid, and extending beyond the anterior border of the muscle. It was not fixed or tender. When it was excised, it was found to be well encapsulated and to extend behind the carotid vessels, the sympathetic trunk being compressed between the tumour and the vessels. Histological examination confirmed the diagnosis of carotid body tumour.

Tuberculous Peritonitis.

Dr. Thomas then presented a female patient, aged thirty-seven years, who had been admitted to hospital, acutely ill, with a firm mass in the abdomen felt above the pubis and arising from the pelvis. Abdominal rigidity and tenderness were present. At laparotomy evidence was found of tuberculous peritonitis, and this was confirmed by the pathologist. Treatment was instituted with streptomycin and penicillin, and the patient made steady progress.

Achalasia of the Œsophagus.

Dr. Thomas's next patient was a male, aged forty-four years, who had had a "stricture" of the œsophagus for fifteen years. He had used an œsophageal dilator satisfactorily during that time, but after two attacks of hæmatemesis had agreed to undergo surgical treatment. The result was satisfactory.

Maldescent of the Testis.

Dr. Thomas's last three patients were boys, aged eight, fifteen and nineteen years respectively, who had undergone Thorek's operation for maldescended testis. They provided illustrations of the operation at various stages.

An Unusual Lesion in the Thigh.

Dr. G. Elliott presented a male patient, aged sixty-five years, who had been admitted to hospital on May 25, 1948. Seven or eight weeks previously he had had a febrile attack, and was examined by a doctor, who thought that he might have osteomyelitis. On the sixth or seventh day of his illness he complained of pains in the thigh. A few days later he noticed a lump, which became larger during the day, but subsided at night. There was later some swelling of the leg. The patient stated that the lump did not pulsate till he was in hospital. On examination of the patient, he was found to have a pulsating tumour about four inches by three inches in extent on the medial side of the middle third of the left thigh. Over this a systolic bruit could be heard. Compression of the femoral artery above the swelling caused the pulsation to cease and the mass to become smaller. It was thought to be an aneurysm of the femoral artery. On May 30, 1948, it was noticed that the tumour was not pulsating and had become somewhat larger and more diffuse. There was a coincident rise of temperature. Pulsation was absent from the femoral and *dorsalis pedis* arteries. The results of the Wassermann and Kline flocculation tests were negative, and a leucocyte count revealed 20,800 leucocytes per cubic millimetre, 84% being polymorphonuclear. On June 9, after the aspiration of old dark blood and pus, the mass was incised. A cavity, which extended down into the femoral canal and contained much old fluid blood and soft dark clot, was drained. Microscopic examination of the aspirated fluid revealed numerous pus cells and streptococci. On culture *Streptococcus viridans* was recovered. Progress was satisfactory till June 25, when further swelling developed, with considerable pitting œdema around the healing wound. More pus and blood clot were evacuated. Treatment with penicillin was instituted, and the inflammation rapidly subsided. The patient was discharged from hospital on July 13, 1948, when pulsation was still absent from the femoral and *dorsalis pedis* arteries.

Obstruction of Common Bile Duct by Adhesions.

Dr. Elliott's second patient was a married woman, who, in June, 1945, had had removed a chronically inflamed gall-bladder containing one stone; the common bile duct was not dilated or fibrosed. Six months later she was readmitted to hospital with nausea, vomiting, flatulence and slight jaundice. At operation the bile duct was opened and some biliary mud washed out. No stones were found. Sounds were passed without obstruction, upwards in the hepatic ducts and downwards to the duodenum. The common duct was drained by catheter for sixteen days. About a year later she complained of colicky upper abdominal pain, nausea but no vomiting, and transient jaundice accompanying the attacks. Relief followed what the patient described as a "trickle" (possibly borborygmi). As she had an incisional hernia, and as a loop of bowel in spasm could be felt on occasions, it was thought that obstruction to the common duct was occurring and was probably due to adhesions. At operation the bile duct was found to be angulated and constricted by adhesions near the duodenal end. The adhesions were separated. The bile duct was opened, no stones or mud were found, and patency was demonstrated by the passage of sounds. The duct was drained by a T-tube, and the patient was discharged from hospital with the tube still in place. It was clamped by day and drained at night until its removal a fortnight later. The patient had since remained well.

Intestinal Obstruction during Pregnancy.

Dr. Elliott then presented a female patient, aged twenty-two years, who had come under his care in January, 1948, when she was six months pregnant. Because of the presence in her urine of numerous pus cells and non-motile organisms as well as some granular and hyaline casts, she was regarded as suffering from severe pyelitis of pregnancy. She complained of upper abdominal pain of a dull aching character associated with nausea and vomiting present for one week. There was a history of past attacks of frequency and scalding of micturition and of swelling of the ankles from time to time. Her bowels had not been opened for five days prior to her admission to hospital. Vomiting continued and became more profuse, and the urinary output declined. When she was examined by Dr. Elliott seven days after her admission to hospital, the patient presented a typical picture of small-bowel obstruction, and the diagnosis was supported by plain X-ray examination of the abdomen. At operation two thick bands constricting the small bowel were found and divided, and an extensive area of adhesion of small bowel to uterus was freed. Premature labour began on the following day, and she was delivered of a stillborn male fœtus. After a somewhat stormy convalescence, in which abdominal distension was a pronounced feature, she recovered.

Traumatic Rupture of the Kidney.

Dr. Elliott's last patient was a boy, aged eight years, who had fallen off a fence on November 7, 1948, and hit his left side on a brick. He developed continuous pain with tenderness and guarding of muscles in the left lumbar region, and, on the following day, hæmaturia. Then the abdomen became distended, but resonant, and his temperature rose. Penicillin was administered, and by November 13 all pain, tenderness and swelling had gone and his temperature was normal. Further hæmaturia occurred, numerous red cells and a moderate number of pus cells being found in the urine on microscopic examination; these had all disappeared by November 30. Excretion pyelography on November 18 revealed a normal right kidney and evidence of a rupture of the left kidney at its lower pole; there was a suggestion that rupture of the left ureter might also have occurred.

(To be continued.)

A meeting of the South Australian Branch of the British Medical Association was held on November 26, 1948, in the Verco Theatre of the Institute of Medical and Veterinary Science, the President, Dr. A. D. Lamphee, in the chair.

Recent Advances in Thoracic Surgery.

Dr. H. D'Arcy Sutherland read a paper entitled "Some Observations on Recent Advances in Thoracic Surgery" (see page 459).

Dr. G. H. Burnell, in opening the discussion, showed four patients treated by pneumonectomy, two for bronchogenic carcinoma, one for bronchiectasis and one for tuberculosis. He urged those responsible for the diagnosis of chest diseases to speed their investigation and, if the indications for surgical intervention were present, to send the patients to the surgeon at the earliest moment rather than to allow them to drift on to inoperability or into a state which rendered surgery more hazardous. He asked Dr. Sutherland to make some further mention of Monaldi drainage technique and bronchial adenoma. He also questioned the advisability of giving X-ray therapy to inoperable subjects. In closing his remarks he paid tribute to his anæsthetist (Dr. Mary Burnell) and reiterated Dr. Sutherland's insistence on team work as a *sine qua non* in successful chest surgery.

Dr. P. S. Messent discussed thoracoplasty, and in so doing stressed the importance of the careful selection of subjects for this measure. He said that the majority of disasters

in thoracoplasties resulted from physicians sending only patients in desperate state for operation when in fact such patients could have been operated on months before in a more suitable phase. He also spoke of some very satisfactory results he had had with chronic lung abscesses treated by lobectomy. He stressed the fact that lobectomy for lung abscess, particularly if unsuccessful drainage had been tried first, was a very difficult procedure; but in his experience the results were so satisfactory that the surgeon should never be deterred from undertaking this line of treatment.

DR. RAY HONE, in making some references to bronchiectasis, emphasized that the physician's approach to the disease should always be one of preparation for surgery. There was no medical treatment which justified the withholding of surgical opinion on any patient, because it was only the trained thoracic surgeon who was in a position to assess those unfortunate people adequately. In speaking of carcinoma of the lung he challenged all clinicians to be constantly aware of the possibility of that disease, because the only hope of cure was in early diagnosis.

DR. I. B. JOSE and DR. JOHN HAYWOOD both spoke of the desirability of the development of an adequately constituted chest unit in South Australia. They considered that at present there was a large gap in the State's medical organization and that the development of such a unit would be of great benefit to the State.

Dr. D'Arcy Sutherland, in reply, said that, with reference to thoracoplasty as a routine measure following pneumonectomy, that might be a theoretical ideal, but pneumonectomies had been performed in large numbers only in the last decade and, as a result, there were insufficient long-term cases on which to base a more definite conclusion. He stated that his own personal feeling was that there was a case to be made out for thoracoplasty following pneumonectomy in non-malignant conditions, but it should be only a one-stage procedure, the ribs being removed down to the previous thoracotomy incision. He outlined the use of Monaldi drainage of large cavities as a preliminary to thoracoplasty, but said that he was not impressed with the technique as originally applied by Professor Monaldi himself. In discussing the treatment of bronchial adenoma he stressed that that condition was always locally malignant and that there were usually bronchiectatic changes in the lung distal to the site of the adenoma, thus excision of the affected lung tissue together with the adenoma was nearly always the treatment of choice. Dr. Sutherland thought that palliative X-ray therapy gave symptomatic relief in some cases of inoperable carcinoma of the lung, so long as the infective element was not too great and so long as too large doses were not employed in an attempt to cure the patient. "Curative" doses had been largely abandoned, because they only made life unbearable for the patient with, in fact, no hope of a resultant cure.

Medical Societies.

THE MEDICAL DEFENCE SOCIETY OF QUEENSLAND.

ANNUAL MEETING.

THE forty-eighth annual meeting of the Medical Defence Society of Queensland was held at British Medical Association House, Wickham Terrace, Brisbane, on Wednesday, March 2, 1949, at 4.30 a.m., Dr. G. W. Macartney, Vice-President, in the chair.

Annual Report.

The annual report was presented and adopted. The report is as follows:

The Council has pleasure in presenting the forty-seventh annual report of the Medical Defence Society of Queensland for the year ended December 31, 1948.

Membership.

The Society has now a membership of 541 as against 526 last year. Of this number six have defaulted in payment of subscription for 1948, and their membership has been suspended.

During the year 35 new members were elected and two were reinstated. Our losses were: deceased 3, struck off 6, retired 3, left State 10.

Obituary.

It is with deep regret that we record the deaths of the following: Dr. D. Gifford Croll, Dr. Paul E. Voss, Dr. F. R. Benson.

Office Bearers and Council, 1948.

President, Dr. Neville G. Sutton; *Vice-President,* Dr. G. W. Macartney; *Honorary Treasurer,* Dr. T. V. Stubbs Brown; *Honorary Secretary,* Dr. R. G. Quinn; Councillors, Dr. A. G Anderson, Dr. John Hardie, Dr. F. W. R. Lukin, Dr. J. G. Wagner, Dr. L. P. Winterbotham.

Medico-Legal.

Three cases which were before the Society last year have been finalized. Two of them were settled out of court, with the approval of the Medical Protection Society, Limited. The third case was defended and a verdict was given in favour of the doctors with costs against the plaintiff (who was not in a financial position to meet them). As one of the doctors concerned is a full-time medical superintendent of a hospital, his costs will, it is hoped, be met by the Hospitals Board. At the time of the alleged negligence in this particular case there was no provision for indemnity insurance on behalf of medical practitioners, as is now in operation.

Three new cases have arisen during the year, one of which appears to have been dropped, as no further action has been taken by the plaintiffs for some considerable time. One case is being proceeded with, and the other case concerns the employee of a doctor, who is covered by indemnity insurance.

Five other matters were brought to the Council by members of the Society, and the necessary legal advice was obtained for them.

Alteration of Articles of Association: Indemnity Insurance.

At the last annual meeting of the Society a resolution was passed recommending to the Council that steps be taken to alter the Articles of Association to increase the annual subscription if it is thought fit. Also that consideration be given to the proposal that all members of the Medical Defence Society of Queensland become members of the Medical Protection Society, Limited.

In dealing with this recommendation, the Council at its meeting held on April 15, 1948, passed the following resolution:

> As from 1st January, 1949, that members of the Society be required to take out indemnity insurance to the extent of not less than £1000 in any one case and £2000 in any one year with the Medical Protection Society, Limited, through the Medical Defence Society of Queensland.

At an extraordinary meeting of members of the Medical Defence Society of Queensland, held on October 27, 1948, of which due notice was given, the following resolution was carried unanimously:

> That the Articles of Association be altered in the manner as set out in the Notice of Motion sent to members under date 27th September, 1948.

This amendment provides that—

> From and including the first day of January, 1949, each member who has not already done so shall effect and each member shall keep in force through the Society as his agent an indemnity with "The Medical Protection Society Limited" (or such other person or corporation as may from time to time be approved by the Council) indemnifying such member against or with regard to actions proceedings claims and demands against such member and in any wise affecting his professional character or interests whether directly or indirectly and also against and with regard to all incidental or consequent losses damages costs charges and expenses and such indemnity shall not be limited to a sum less than one thousand pounds sterling in any one case or two thousand pounds sterling in any one year.

Income Tax: Subscriptions.

A ruling has been obtained from the Commissioner of Taxes to the effect that payments to the Medical Protection Society, Limited, and subscriptions to the Medical Defence Society of Queensland, not exceeding £10 10s. *per annum,* are regarded as allowable deductions for income tax purposes.

Finance.

The total assets of the Medical Defence Society of Queensland amount to £7311 11s. 8d., which includes £71 5s. 9d. repre-

senting indemnity insurance subscriptions to be remitted to London. Of this amount £6563 18s. is invested in Commonwealth Government bonds and stock. On the year's operations there was a deficit of £426 2s. 10d., due to abnormal legal expenses, which amounted to £895 18s. 6d. General expenses totalled £99 3s. 2d. Income was derived as follows: annual subscriptions and entrance fees, £321 0s. 7d.; Commonwealth bonds and stock interest, £235 17s. 6d.; Commonwealth Savings Bank interest, £12 0s. 9d.

Neville G. Sutton,
President.

Balance Sheet and Financial Statement.

The balance sheet and financial statement for the year ended December 31, 1948, was taken as read and adopted.

Election of Councillors.

Dr. R. G. Quinn was reelected to the Council. Dr. F. Garrett Scoles, Dr. Athol Quayle and Dr. H. Masel were elected to fill the vacancies on the Council. It was reported that Dr. H. G. Anderson, Dr. L. P. Winterbotham and Dr. John Hardie did not seek reelection. It was resolved that letters of thanks be sent to them expressing appreciation of their services to the Society in the past.

Naval, Military and Air Force.

APPOINTMENTS.

The undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 19, of March 3, 1949.

Naval Forces of the Commonwealth.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointments.—Geoffrey Derek Banyard is appointed Surgeon Lieutenant (for Short Service), dated 6th December, 1948. Patrick Millar Littlejohn, Harold Marcus Rhys James and William McLaren Thomson are appointed Surgeon Lieutenants (for Short Service), dated 1st January, 1949. Ernest Brian Busfield and Alfred Gordon Robertson are appointed Surgeon Lieutenants (D) (for Short Service), dated 20th December, 1948, and 25th January, 1949, respectively.

Termination of Appointment.—The appointment of Norman Thomson Smith as Surgeon Lieutenant (for Short Service) is terminated, dated 13th December, 1948.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Volunteer Reserve.

Peter Dudley Blaxland is appointed Surgeon Lieutenant with seniority in rank of 30th August, 1943, dated 3rd August, 1948.

Australian Military Forces.

Interim Army.

Australian Army Medical Corps (Medical).

N73138 Lieutenant-Colonel (Temporary Colonel) A. M. McIntosh relinquishes the appointment of Deputy Director of Medical Services, Headquarters Eastern Command, and is attached Headquarters Eastern Command, 1st July, 1948.

To be Temporary Majors.—Captains NX506207 P. A. McReady, 4th September, 1948, and SX700004 R. N. McCann, 9th October, 1948.

To be Captain, 20th October, 1948.—VX700067 George Pentland Cromie.

Active Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—The following officers are appointed from the Reserve of Officers with Regimental Seniority in accordance with Army Seniority, 1st May, 1948: 1/39015 Major E. P. Row and 1/39016 Captain D. K. Lehmann.

Australian Army Medical Corps (Medical).—1/43710 Colonel K. B. Fraser, E.D., from Deputy Director of Medical Services (Part-Time Duty), Headquarters, Northern Command, is appointed Deputy Director of Medical Services, Headquarters Northern Command, 1st July, 1948. The following officers are appointed from the Reserve of Officers, with Regimental Seniority in accordance with Army Seniority, 1st May, 1948: Captains 1/32022 K. H. Hooper and 1/28390 I. H. Chenoweth. *To be Captain (provisionally), 19th October,* 1948.—1/39022 Peter James Landy.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/100752 Captain R. D. Rothfield is appointed from the Reserve of Officers, with Regimental Seniority in accordance with Army Seniority, 1st May, 1948. *To be Captain (provisionally), 15th November,* 1948.—2/127803 Russell Markham O'Donohue.

Australian Army Medical Corps (Medical).—2/142865 Captain T. H. Lee is appointed from the Reserve of Officers with Regimental Seniority in accordance with Army Seniority, 1st May, 1948. 2/50001 Lieutenant-Colonel (Honorary Colonel) S. H. Lovell relinquishes the honorary rank of Colonel and is promoted to the temporary rank of Colonel, 1st April, 1948.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/50056 Lieutenant-Colonel J. G. G. White, O.B.E., is appointed from the Reserve of Officers, 1st April, 1948, with Regimental Seniority in accordance with Army Seniority, is appointed Deputy Director-General of Medical Services, A Branch, Army Headquarters, and is promoted to the temporary rank of Colonel, 14th December, 1948.

Australian Army Medical Corps (Medical).—3/50011 Lieutenant-Colonel (Temporary Colonel) C. W. Ross from Chairman, Medical Equipment Advisory Committee (part-time duty), Director of Medical Services, A Branch, Army Headquarters, is appointed Colonel Advisory Committee Medical Equipment, Director of Medical Services, A Branch, Army Headquarters, 1st July, 1948. 3/123264 Captain B. Clerehan is appointed from the Retired List (3rd Military District), 1st May, 1948. *To be Captains (provisionally), 15th November,* 1948.—3/101809 John Kevin Clarebrough (with Regimental Seniority next after 3/87148 Captain (provisionally) W. L. Jenkins) and 3/101808 Rowland Eric Seal (with Regimental Seniority next after 3/101809 Captain (provisionally) J. K. Clarebrough).

Southern Command: Fourth Military District.

Australian Army Medical Corps (Medical).—4/31935 Captain D. F. Hannon is appointed from the Reserve of Officers with Regimental Seniority in accordance with Army Seniority, 1st May, 1948.

Southern Command: Sixth Military District.

Royal Australian Army Medical Corps (Medical): To be Captains (Provisionally), 3rd December, 1946.—6/15316 Donald Edmund Anderson, 6/9022 John Oliver and 6/15206 David John Walters.

Australian Army Medical Corps (Medical).—6/15202 Major (Temporary Lieutenant-Colonel) J. A. Gollan is appointed Deputy Director of Medical Services, Headquarters, 6th Military District, 1st July, 1948, and is promoted to the temporary rank of Colonel, 15th October, 1948. 6/15311 Major P. Braithwaite is appointed from the Reserve of Officers, 1st May, 1948, is appointed to command 12th Field Ambulance, 15th September, 1948, and is promoted to the temporary rank of Lieutenant-Colonel, 15th October, 1948. 6/15312 Captain N. D. G. Abbott is appointed from the Reserve of Officers, with Regimental Seniority in accordance with Army Seniority, and is promoted to the temporary rank of Major, 10th July, 1948. 6/15314 Honorary Captain L. W. Knight is appointed from the Reserve of Officers, relinquishes the honorary rank of Captain and is appointed Captain (provisionally), 1st May, 1948.

Western Command: Fifth Military District.

Australian Army Medical Corps (Medical).—The following officers are appointed from the Reserve of Officers, with Regimental Seniority in accordance with Army Seniority, 1st May, 1948: 5/26393 Major L. P. Gray, and Captains (Honorary Majors) 5/26392 A. J. King and 5/26394 H. J. Rowe. *To be Captain (provisionally), 15th November,* 1948.—5/20781 Clement Vernon Love. *To be Temporary Major, 1st May,* 1948.—5/26392 Captain (Honorary Major) A. J. King and relinquishes the honorary rank of Major.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District: To be Honorary Captain, 24th November, 1948.—Charles William Leece. *To be Honorary Captain, 20th December,* 1948: Thomas Joseph Ryan.

Australian Army Medical Corps.

3rd Military District: To be Honorary Captains.—Ralph Reginald Clark and Thomas Henry Hurley, 9th November, 1948, and Christopher Bruce Bailey and Ross Wharton Webster, 15th November, 1948.

4th Military District: To be Honorary Captains, 15th November, 1948.—Donald Douglas Beard, Brian Leslie Cornish, Hugh Matheson Douglas, Geoffrey Thomas Ey, Donald Barton McLeay and Geoffrey Lloyd Mellor.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

General Revision Course.

THE annual two-week general revision course will begin in Sydney on May 23, 1949, under the supervision of Dr. Frank L. Ritchie.

Considerable changes are being made to the programme of this course. The time devoted to practical medical subjects has been increased, technical aspects of surgery and other subjects have been decreased and lectures in dermatology have been added. In addition to this, lecturers from other States include Dr. F. Ray Hone, Dr. J. D. Hayden and Dr. Ian J. Wood.

Increased attention is being paid to the social aspects of the course so that in most cases both members and their wives may be able to participate. Social functions include a cocktail party, a theatre party, a dinner and the annual competition for the post-graduate golf cup.

The fee for attendance will be £5 5s., and a programme will be available shortly. Those wishing to enrol are requested to communicate with the Course Secretary, 131 Macquarie Street, Sydney.

Gynæcology and Obstetrics.

The Post-Graduate Committee in Medicine in the University of Sydney announces that from April 1, 1949, post-graduate residencies can be arranged at the Royal Hospital for Women, Paddington, for short periods. Fees are £5 5s. per week, including board and residence, and bookings must be made with the Course Secretary of the Committee, 131 Macquarie Street, Sydney. Telephones: BW 7483-BU 5238.

Correspondence.

THE GOVERNMENT AND THE MEDICAL PROFESSION.

SIR: Now that the Government has bared its fangs to show the teeth of dictatorial coercion to an ancient and noble profession, is it not high time that we called trained forces to our aid in a full-scale effort to denounce this evil thing?

Hitherto, we have stated our case honestly and with dignity to Ministers under the justifiable impression that people in their position (no matter what their politics!) are genuinely attempting to achieve some high-minded design. However, it has now become obvious that they have nothing in view for the good of the people—they merely have a ferocious lust for power, a lust which includes departmental control over the medical profession as a whole. It seems clear that it is just such control at which they have been aiming right throughout their discussions with our Council.

In my opinion, and in the opinion of others to whom I have spoken, we should now mobilize and be led by professional advice.

A campaign conducted by an advertising or publicity agency of repute, with legal guidance in the background, surely would go a long way. This fight must no longer be waged by doctors alone against the socialists—it must be brought before the people in language and ways which they will clearly understand. It is the people who must decide this issue and nobody else; but so long as the statements of trained politicians are allowed to dominate the pages of the Press to the exclusion of the views of the doctors, how can the people possibly realize the drastic implications of this governmental coercion? Such a campaign will cost money. Well then, let us raise adequate money and more. The Federal Independence Fund is a start; let us have a capital levy as well. We must win this fight against tyranny—and if we are genuinely in favour of winning, we must be prepared to pay for winning. A victory in this sphere might help turn the tide against socialism in general and its attendant evils—we could wield enormous political influence if we only knew it. For goodness' sake, then, let us wield it now before the chance has gone forever.

Yours, etc.,

J. L. DOWLING.

149 Macquarie Street,
Sydney,
New South Wales.
March 18, 1949.

[The constitution of the British Medical Association will not allow a levy to be made among its members.—EDITOR.]

MAGNESIUM IN DYSMENORRHŒA.

SIR: I thank "Woman Doctor" (THE MEDICAL JOURNAL OF AUSTRALIA, February 19, 1949) for her observations on "Magnesium in Dysmenorrhœa". It was for that reason I published a small series of cases. In personal communications, I must say most doctors have so far observed quite beneficial results, fairly comparable in percentages to those in my paper.

I have not studied the seasonal and climatic variations in severity, but will follow this useful lead.

Regarding adequate doses, as stated in my article, I now give two or three doses per day for three or two days premenstrual and menstrual if a single daily dose does not suffice.

I have not facilities for checking the magnesium content of muscle tissue and the blood plasma level. One has the analogy that ingestion of dextrose does not raise the blood sugar level, but presumably the sugar is absorbed and performs its function.

How to evaluate the psychological factor in statistics is a difficult matter for which I apologized—of course conversely, if it is such a big factor in dysmenorrhœa, has "Woman Doctor" consulted a psychologist?

Yours, etc.,

W. J. RAWLINGS.

12 Collins Street,
Melbourne, C.1,
March 7, 1949.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Hill, William Howard, M.B., B.Ch., B.O., 1932 (Queen's Univ., Belfast), Longford, Tasmania.

Kerridge, Gordon, M.B., B.S., 1943 (Univ. Sydney), Royal Hobart Hospital, Hobart.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Acts, 1939 to 1948*, of Queensland, as duly qualified medical practitioners:

Toakley, James Geoffrey, M.B., B.S., 1946 (Univ. Sydney), Mater Hospital, South Brisbane.

Westaway, Leslie, M.B., B.S., 1947 (Univ. Sydney), Rockhampton Hospital, Rockhampton.

Kenny, Geoffrey Charles Treadgold, M.B., B.S., 1949 (Univ. Queensland), "Nirvana", Norman Court, East Brisbane, Brisbane.

Pearson, Kenneth Malcolm, M.B., B.S., 1949 (Univ. Queensland), 89 Chatsworth Road, Greenslopes, Brisbane.

Webb, John Vincent, M.B., B.S., 1949 (Univ. Queensland), Abbotsleigh Road, Holland Park, Brisbane.

Bagley, George Joseph, L.R.C.P., L.R.C.S., 1948 (Edinburgh), L.R.F.P. and S. (Glasgow), corner Junction and Zillman Roads, Hendra, Brisbane.

Dan, Raymond Victor, M.B., B.S., 1949 (Univ. Sydney), General Hospital, Brisbane.

Macfarlane, Walter Victor, M.B., B.S., 1944 (Univ. New Zealand), c.o. Department of Physiology, University of Queensland.

Toyne, Albert Howard, M.B., B.S., 1944 (Univ. Melbourne), Cooroy.

Congresses.

FIRST INTERNATIONAL CONGRESS OF BIOCHEMISTRY.

THE First International Congress of Biochemistry will be held at Cambridge from August 19 to 25, 1949. The congress will be divided into twelve sections. The chairman is H. J. Bunker, M.A., and the secretary is P. W. Brian, Ph.D., Butterwick Research Laboratories, The Frythe, Welwyn, Herts.

Obituary.

SIDNEY VALENTINE SEWELL.

WE regret to announce the death of Sir Sidney Valentine Sewell, which occurred on March 12, 1949, at Melbourne. This name was incorrectly stated in the issue of March 26 as Sidney Arnold Sewell.

JOHN HARVEY JOHNSTON.

WE regret to announce the death of Dr. John Harvey Johnston, which occurred on March 17, 1949, at Toorak Gardens, South Australia.

Notice.

S. E. JONES MEMORIAL FUND.

THE following subscriptions to the S. E. Jones Memorial Fund have been received up to March 2, 1949.

	£	s.	d.
Previously acknowledged	228	10	0
Dr. W. R. Page, Dr. S. W. King, Dr. C. MacMahon, Dr. S. McLennon (£5 5s. each)	21	0	0
Dr. A. T. Edwards	5	0	0
Dr. I. Sebire	2	2	0
Dr. S. G. Sandes	2	2	0
Dr. H. M. North	2	2	0
Dr. B. Joyce	1	2	0
Total	£261	18	0

Cheques made payable to the S. E. Jones Memorial Fund may be sent to the Medical Secretary, New South Wales Branch of the British Medical Association, 135 Macquarie Street, Sydney.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Fewtrell, Ralph Douglas, M.B., B.S., 1948 (Univ. Sydney), Flat 1, 109 Ben Boyd Road, Neutral Bay.

Burfitt, Barbara Joyce, M.B., B.S., 1940 (Univ. Sydney), Yeoval, New South Wales.

Simpson, John Heaton, M.B., B.S., 1937 (Univ. Sydney), 101 Willoughby Road, Crow's Nest.

McLachlan, Colin Stewart, M.B., B.S., 1948 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Lister, Harvey Alexander John, M.B., B.S., 1944 (Univ. Sydney), 13 Union Road Auburn.

Diary for the Month.

APRIL 5.—New South Wales Branch, B.M.A.: Council Meeting.
APRIL 6.—Victorian Branch, B.M.A.: Branch Meeting.
APRIL 6.—Western Australian Branch, B.M.A.: Council Meeting.
APRIL 7.—South Australian Branch, B.M.A.: Council Meeting.
APRIL 8.—Queensland Branch, B.M.A.: Council Meeting.
APRIL 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APRIL 12.—New South Wales Branch, B.M.A.: Organization and Science Committee.
APRIL 14.—Victorian Branch, B.M.A.: Organization Subcommittee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA *alone*, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £3 *per annum* within Australia and the British Commonwealth of Nations, and £4 10s. *per annum* within America and foreign countries, payable in advance.